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COMPENDIUM OF REGIONAL DIAGNOSIS IN AFFECTIONS OF THE BRAIN AND SPINAL CORD

A CONCISE INTRODUCTION TO THE PRINCIPLES OF CLINICAL LOCALIZATION IN DISEASES AND INJURIES OF THE CENTRAL NERVOUS SYSTEM

BY

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TRANSLATED FROM THE SIXTH GERMAN EDITION BY
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THIRD EDITION
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PREFACE TO THE SIXTH EDITION

As, since the appearance of the fifth edition, the subject with which I am dealing has not been enriched by any notable acquisitions, extensive alterations in the text have not been necessary. Special attention has therefore been given to a thorough revision of the illustrations. Diagrams which could no longer be regarded as abreast of the present state of our knowledge have been replaced by others better and more forcible. Further, thanks to the willing collaboration of my publishers, many of the illustrations have undergone a thorough technical revision, and thereby the serviceableness of my Compendium has been very happily furthered.

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PREFACE TO THE FIFTH EDITION

A COMPARISON of this latest edition of my Compendium with its original form will manifest in striking fashion the extraordinary enrichment and development of our knowledge of cerebral and spinal localization which a comparatively short space of time has brought about. The years of the world war, too, which laid fallow the field of research in so many other branches of knowledge, put at the disposal of the diagnostician in this field an almost overwhelming mass of observations and experiences. I have endeavoured to utilize, side by side with the results of my own observations of wounded men interned in Switzerland, communications on war neurology from the belligerent countries, giving, it need hardly be said, as careful consideration to the work of my *confrères* as to my own. With one qualification, however. For the recent luxuriant growth of theoretical speculation concerning the fundamental conceptions with which regional diagnosis must necessarily work—conceptions such as centre, inhibition, loss of function, irritative phenomena, etc.—there is, in my opinion, no room within the framework of a work whose general direction should remain as it was laid down in the Introduction to the first edition. For this reason I have declined to be enticed from the firm ground of a statement of that which is utilizable in diagnosis on to the boundless ocean of hypothesis. Even for a special domain of physio-pathology such as ours the statement of the physiologist Gley applies, that theories are powerfully attractive to most men because they present the philosophy, often indeed the poetry, of science; but that the accumulation of observation and experience creates values which endure, while the most beautiful hypotheses are subject to endless change. Without such refusal it would have been impossible to accommodate this book to the yearly growing mass of facts with an addition of no more than fifty pages, and so to preserve its character of a compendium.

PREFACE TO THE FIRST EDITION

While delivering a course of lectures on regional diagnosis to medical men during the winter session 1907-08, I was repeatedly urged by members of my audience to publish a book which should give in a concise form, and one suited to readers who are not neurologists, the substance of my lectures. I was unable to fall in with the suggestion. There is no lack of excellent textbooks dealing with the subject. A short, practical vade-mecum of a didactic character has not, however, to the best of my knowledge, been, up to the present, available, and yet it seemed probable that such a work would be by no means unwelcome to the physician or surgeon called upon to localize a pathological process affecting the central nervous system. It is from this point of view that the present Compendium took its origin. Its chief task is to give a presentation of a subject generally reputed difficult and complicated, which shall be as clear and simple as the author can make it, while not failing to be accurate and comprehensive, and which shall furnish, even to those who have special knowledge of neurology, an easily and quickly consulted work of reference. In such a scheme there is no room either for the putting forward of elaborate theories or the discussion of controverted points. At the same time I have nowhere shirked the attempt to give a scientific explanation of clinical facts. In the main, only such material has been put forward as is to be regarded as, in the present state of our knowledge, firmly established. Where this is not the case, attention is specially called to the fact that our knowledge on the point dealt with is still incomplete. The names of authors are only given where they have become part of neurological nomenclature—*e.g.*, Brown-Séquard's symptom-complex, Westphal-Edinger nucleus, etc. Quotation from other authors is limited to the few occasions on which I have directly borrowed a table or a diagram. The illustrations are those which served me as wall diagrams in my lectures. This must be my apology for their frequently simple and sketchy character, which, however, is more conducive to a rapid apprehension and comprehension of the matter put forward than would be the case with a less diagrammatic form of illustration.

ROBERT BING.

Basle, 1909.

INTRODUCTION

The object of that branch of the science of neurology denominated "regional diagnosis" is, by inference from the clinical phenomena called forth by a circumscribed lesion of the central nervous system, to localize that lesion —that is to say, to determine (without, in the first place, any reference to its special nature) its exact anatomical situation.

Regional diagnosis bases its conclusions on the observation of disease symptoms arising from the injury or destruction of separate structures forming part of the central nervous system. Inasmuch as all these structures (nerve nuclei, nerve tracts, etc.) have clearly defined situations in the brain or the spinal cord, it is possible to deduce the situation of a lesion from the combination of irritative and paralytic symptoms found to be present in the given case. Regional diagnosis, then, borrows its tools alike from anatomy and from physiology. In essence, the science of clinical localization is a collation and comparison of data furnished by those two sciences. It is, in fact, fundamentally the applied anatomy and physiology of the central nervous system. Its practical importance is immense. In all lesions of the nervous system the prognosis depends quite as much on the situation as on the specific anatomico-pathological nature of the lesion, and, where surgical interference is in question, regional diagnosis is, of course, of equally vital importance.

TRANSLATOR'S NOTE

In a few cases references in German form part of the plates. As their removal and replacement by English equivalents involved difficulties and considerable expense, translations have been given in the index.

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INTRODUCTION TO AMERICAN EDITION

It is a privilege to write a few words in introduction to Professor Bing's Compendium of Regional Diagnosis and to its adequate translation into English and to its translator, F. S. Arnold. This book has reached its sixth German and its third English edition. This means that it has been of assistance to many medical students, to some neurologists, and, let it be hoped, to a few internists and surgeons.

Neurology is a difficult subject, a complex and intricate medical specialty. It is made more difficult than need be because it is so often approached without the necessary anatomical and physiological knowledge. To understand diseases of the nervous system and to cultivate an interest in its problems it is essential to know and to know accurately something about the relations which the nervous system bears to other anatomical and physiological systems. There is need to cultivate a feeling and a realization of the topography of muscle, segment, bone and nerve supply in its proper functional relationship. This knowledge must not be made up of isolated strings of facts, terms, and classifications, but should be in the nature of cross-sections and integrations of living and properly functioning parts. It is the organism as a whole, the total person, in its neurological phase that it is essential to know about. It is this sort of thing that Bing's compendium attempts to supply, and that it does this well is testified by its increasing popularity, of which this new edition is a compelling proof. As an introduction to the study of organic diseases of the nervous system, the method here pursued is admirable. The coordination between anatomy and function is beautifully done. The diagrams and figures are carefully chosen. A book of this kind is not perhaps interesting of itself. It is obviously not meant to be studied by itself. It should be used as a guide and companion to clinical descriptions and to the actual scrutiny of cases of nervous diseases. It should share intimately the vital contacts with the living reality of disease. The information contained in this compendium is the groundwork and the essential basis towards the understanding of the nervous system and its maladies. Its facts are the hard won facts of anatomy-physiology and the close relationship of one with the other. These must be known before they can be used as instruments through which the human being with a nervous disease can be understood, diagnosed and treated.

It is fortunate for the student that this information is given to him in so clear, precise and instructive fashion. It is well to know that books with this purpose form the solid groundwork upon which increased knowledge of the nervous system can be built up. From physiology and anatomy in its broadest sense will come an under-

standing of the infinitely more complex states of disordered function, the neuroses and psychoses which unfortunately cannot at the present time have such a compendium as a guide. It is to be hoped that some time in the future a similar compendium will be available for the study of these situations and that it will have something of the same precision, accuracy, and definiteness so characteristic of this book.

It is a pleasure, then, to set down in its early pages a word of praise and commendation to the effort so valiantly made to give the inquiring and curious student a guide that he will cheerfully follow into the intricate pathways of neurological diagnosis.

SIDNEY I. SCHWAB.

DIVISION I

REGIONAL DIAGNOSIS OF LESIONS OF THE SPINAL CORD

DIVISION I

REGIONAL DIAGNOSIS OF LESIONS OF THE SPINAL CORD

In attempting to localize a focus of disease or lesion of any kind in the spinal cord, two points of capital importance have to be kept in view, owing to the cylindrical or cordlike form of the organ. In the first place, we must determine whether the lesion is central or peripheral, anterior or posterior, right or left, and over how large an area it has interrupted the spinal nerve channels and destroyed the spinal centres. In the second place, we wish to know at what level, at what point of its extent, the cord is affected by injury or disease. We may speak, therefore, of a " transverse " or systemic, and a " longitudinal " or segmental, diagnosis of spinal affections, and will proceed now to the consideration of their foundation principles.

A. TRANSVERSE OR SYSTEMIC DIAGNOSIS.

It is necessary to begin with an outline of the anatomical conditions, which, however, may be very simple and brief, seeing that our point of view is to be purely clinical and utilitarian. A knowledge of the anatomy of the spinal cord, and especially of the anatomical nomenclature of its parts, is, of course, assumed.

The broad division into white and grey matter corresponds to a finer division into conducting nerve channels and nerve-cell network. We must realize, therefore, in the first place, the position and course of the former, and the grouping and division of the latter (Chapter I.). If we then bring into relation with the foregoing a survey of the normal functions of these elements so far as they are known to us (Chapter II.), certain regional diagnostic conclusions are seen to follow naturally from disturbance or inhibition of those functions (Chapter III.).

CHAPTER I.

I. THE SPINAL TRACTS.

In works on descriptive anatomy the spinal tracts are divided into " long " or " short." From a clinical point of view, we must select another principle of division, and group the tracts under the following headings:

(*a*) *Tracts whose cells of origin lie outside the spinal cord.*

These tracts merely pass through the cord; taking their origin elsewhere—in the brain, for example, or in the spinal ganglia. These tracts are liable to destruction, as the result, not only of primary disease of the cord, but also of cerebral and ganglionic lesions—in accordance with Waller's law: "A nerve fibre can only maintain its anatomical and physiological integrity so long as it remains in undisturbed continuity with a healthy nerve cell." These tracts are called *exogenous*.

(*b*) *Tracts whose cells of origin lie within the spinal cord.*

To bring about destruction of these tracts throughout their whole extent, a lesion of the spinal cord itself is required, and, further, of the grey matter of the cord, the exclusive seat of the spinal nerve cells. These tracts are called *endogenous*.

A. EXOGENOUS TRACTS.

I. Descending or Centrifugal (see Fig. 1).

1. **Tracts descending from the Cerebral Cortex = Fasciculi Cortico-Spinales = Pyramid Tracts.**

The cells of origin of these tracts lie in the motor area of the cortex cerebri; the tracts themselves pass, their fibres converging as they go, through the substance of the parietal lobe to the internal capsule, traverse the posterior limb of the latter, and continue their further course through the ventral portions of the pedunculi and the pons to the medulla oblongata. Here the fibres divide into two systems:

(α) **The Lateral Pyramid Tracts = Fasciculus Cortico-Spinalis Lateralis.**

The greater number of the cortico-spinal fibres cross from the pyramid of the medulla oblongata to the opposite side of the spinal cord at the upper limit of the latter, and pass downwards in the lateral columns of the cord, till their neurons find their terminal divisions in the cells of the *anterior horn*.

(β) **The Anterior Pyramid Tracts — Fasciculus Cortico-Spinalis Anterior.**

A smaller number of the cortico-spinal fibres pass downwards without crossing at the upper limit of the cord through the anterior columns. These also find their termination in the cells of the anterior horn, but before doing so cross from one side to the other of the cord by way of the anterior commissure, thus undergoing a delayed, preterminal decussation.

2. **Tracts descending from the Lower Brain Centres = Fasciculi Subcortico-Spinales.**

Under this head I include the following:

(α) **Tractus Rubro-Spinalis = Monakow's Bundle.**

This springs from the red nucleus of the tegmentum cruris cerebri. It undergoes decussation soon after its origin, and passes downwards in the lateral column of the cord.

(β) **Tractus Tecto-Spinalis.**

Arises from the roof of the mid-brain (*Tectum opticum*), decussates with its fellow beneath the aqueduct of Sylvius, and, passing through the pons and medulla close to the middle line, descends in the ventral column of the spinal cord.

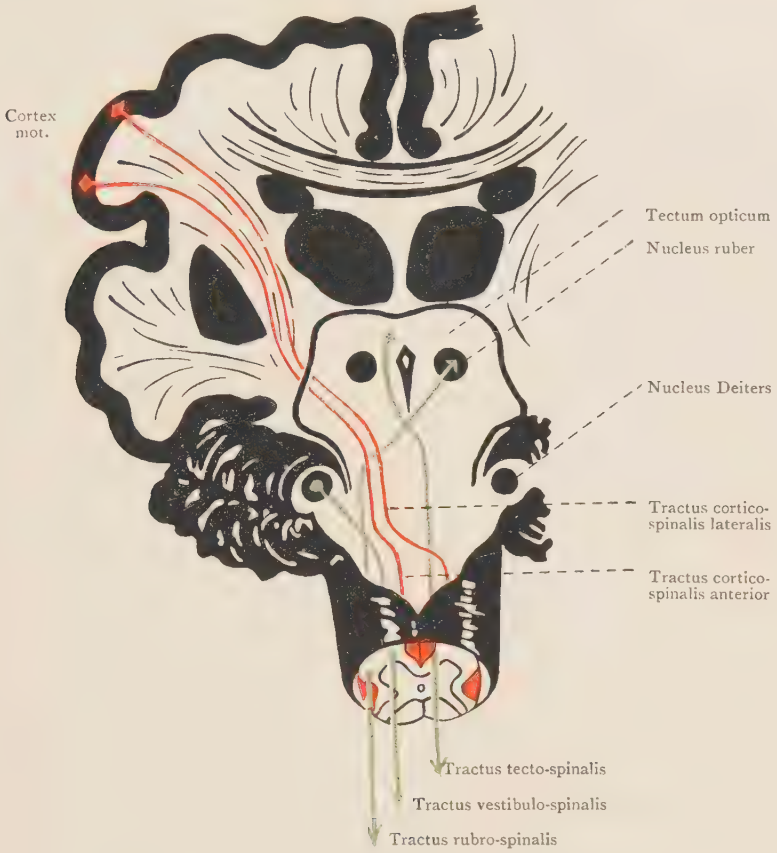


FIG. 1.—DESCENDING EXOGENOUS SPINAL TRACTS.
Red : Tractus cortico-spinales = chief motor tracts.
Green : Tractus subcortico-spinales = secondary motor tracts.

(γ) **Tractus Vestibulo-Spinalis.**

Is derived from the nucleus of Deiters in the medulla oblongata, which belongs to the system of the vestibular nerve. Passes downwards, uncrossed, in the ventral column of the cord, and ends among the cells of the ventral horn, the probable seat of termination of the subcortico-spinal tracts in general.

II. Ascending or Centripetal Tracts.

The ascending exogenous tracts have their cells of origin in the spinal ganglia. These cells are connected peripherally by means of fibres running in the

nerve trunks, with the various peripheral organs, "nerve endings," "terminal corpuscles," "end-bulbs," "tactile corpuscles," "Pacinian bodies," and so on, found in the skin, the mucous membranes, the mesentery, the articular surfaces, and elsewhere. Centrally the fibres from the cells of the spinal ganglia pass by

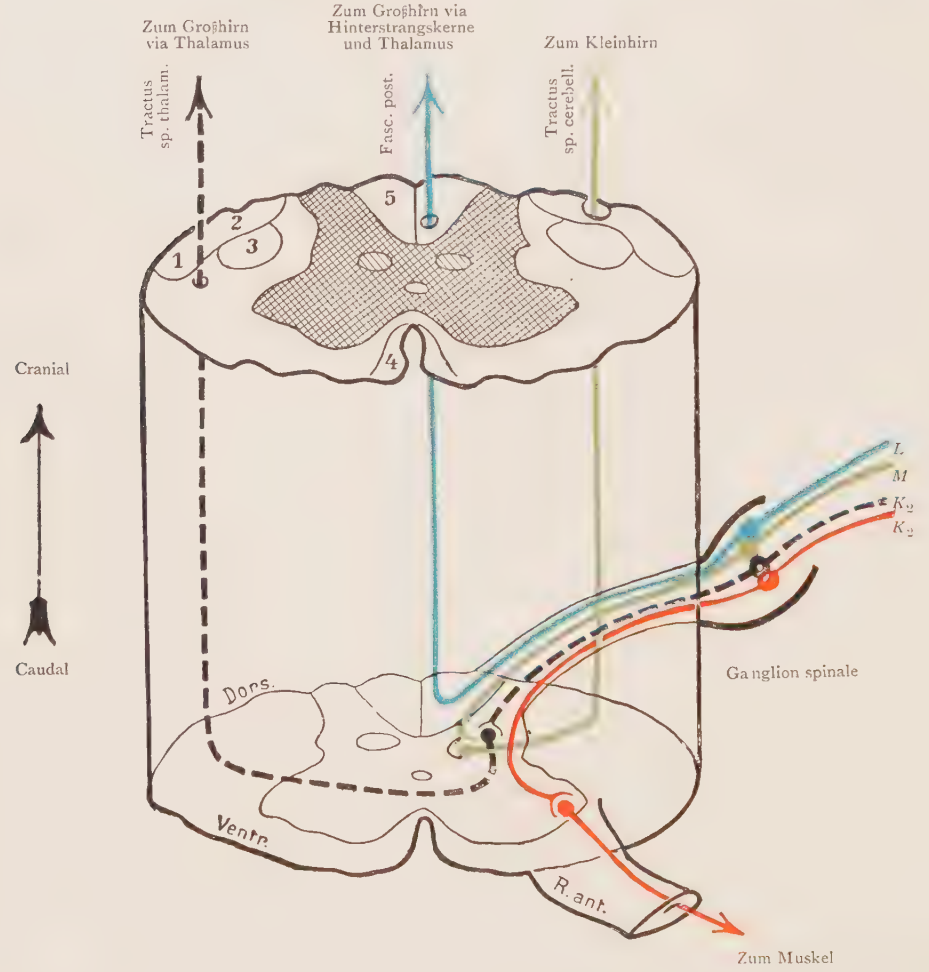


FIG. 2.—THE VARIOUS GROUPS OF POSTERIOR ROOT FIBRES AND THEIR CONTINUATIONS WITHIN THE SPINAL CORD.

way of the posterior roots into the spinal cord. These fibres may be divided, according to their further course within the cord, into three sections (Fig. 2).

(α) **Short Fibres of the Posterior Roots.**

These pass directly through the marginal zone of the posterior horn into the grey matter, where they end by arborizing among the cells (1) of the posterior, (2) of the anterior horn, on the same side of the cord.

(β) **Medium Fibres of the Posterior Roots.**

These pass through the segment of entry into the posterior columns, and from thence into the substance of the dorsal horn of the same side, ending in the *column of Clarke.*

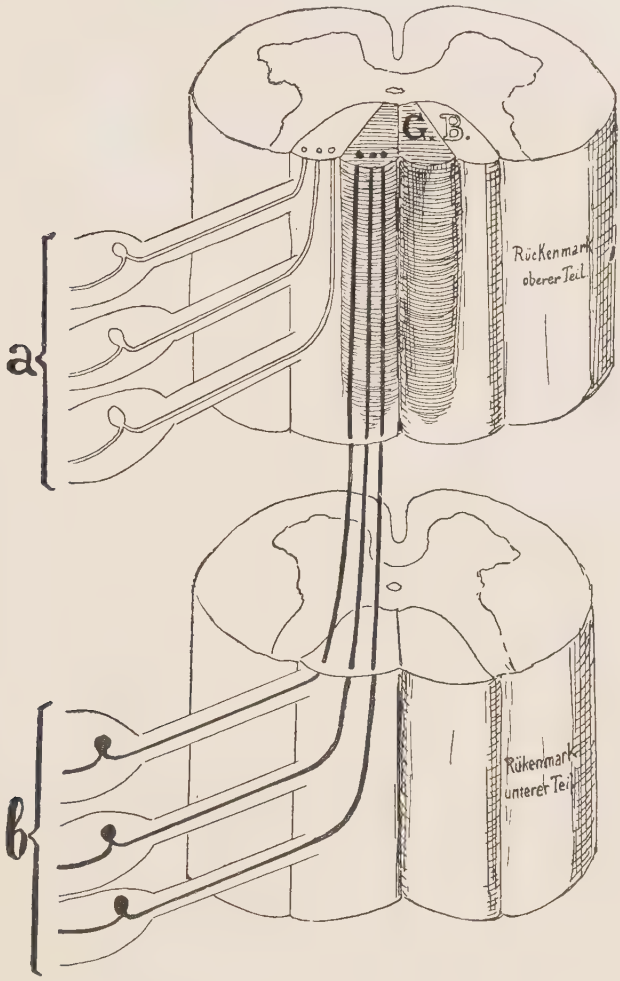


Fig. 3.—Formation of the Posterior Columns.

G=Column of Goll (funiculus gracilis).
B=Column of Burdach (funiculus cuneatus).
a=Long posterior root fibres from the upper half of the body.
b=Long posterior root fibres from the lower half of the body.

(γ) **Long Fibres of the Posterior Roots.**

These also pass, through the segment of entry, into the posterior columns, but, in these, pursue their further course in the direction of the brain, and terminate in certain nuclei in the medulla, *the nuclei of the dorsal columns.*

The two following anatomical peculiarities of these fibres of the posterior columns have also pathological significance:

(1) As the fibres from a particular posterior root pass upwards in the dorsal columns, they are pressed more and more towards the middle line by fibres of the same kind, entering through the zone of entry of higher segments. In consequence of this, in a transverse section of the cervical cord, the fibres from the sacral roots lie nearest the septum; next to them, a little more externally, are ranged the lumbar fibres, then the thoracic, and, finally, adjacent to the dorsal horns, the fibres from the cervical roots.

In this manner there is brought about in the cervical cord a macroscopic separation between the long posterior root fibres from the lower half of the body (from the fourth dorsal segment downwards), which are united in the column of Goll, and those from the upper half of the body (from the fourth dorsal segment upwards), which form the column of Burdach. Fig. 3 gives a diagrammatic representation of this distribution of fibres.

(2) Each long posterior root fibre, before commencing its passage brainwards in the dorsal column, sends off a branch which passes for some distance in a downward or caudal direction, also in the dorsal column. These descending elements of the posterior root system unite to form a special tract, the so-called *comma tract of Schultze*. The comma tract, therefore, contains at any given level only fibres which have entered the cord at a higher level. For instance, in the upper lumbar segments the comma tract contains no lumbar fibres, but only fibres which have entered higher up, and belong, in the given case, to certain dorsal segments.

B. ENDOGENOUS TRACTS.

I. Centripetal Fibres of the Second Order (*vide* Fig. 2).

Under this head we include those tracts which connect with elements of the posterior root system that have already terminated in the cord, and provide for the further conduction, towards the higher centres, of impulses received through those elements.

They are—

1. **The Spino-Cerebellar Tracts.**

(α) **The Dorsal Spino-Cerebellar Tract.**

These fibres arise in the cells of the column of Clarke, pass upwards in the dorsal periphery of the lateral column of the same side to the medulla, and from thence by the restiform body to the cerebellum, ending in the vermis. They form the connecting neurons between the above-mentioned medium fibres of the posterior root system and the higher centres.

(β) **The Tract of Gowers, or Ventral Spino-Cerebellar Tract.**

The cells of origin of this tract are situated in the lateral portions of the substance of the ventral horn. Its fibres pass upwards in the ventral periphery of the lateral column, partly on the same side of the cord, and partly on the opposite side after crossing the anterior commissure. After traversing the medulla and pons, they enter the cerebellum by way of the superior peduncle, along which they pass to the vermis. They form the connecting neurons for the short fibres of the posterior root system. These fibres are not given in Fig. 2.

2. **The Spino-Thalamic Tract.**

This tract also arises from cells in which terminate short fibres from the posterior roots. These cells, however, are situated in the dorsal horn. The fibres of the spino-thalamic tract decussate, as do some fibres of the tract of Gowers, by way of the anterior commissure, and pass upwards in the lateral column, terminating, however, not in the cerebellum, but in the optic thalamus.

II. The So-called Intersegmental or Associating Tracts.

These tracts serve to connect the grey matter of successive segments of the cord. The names "proprio-spinal," "intrinsic," or "spino-spinal" are given to their fibres. They are found—

(1) **In the Ventro-Lateral Column.**

(2) **In the Dorsal Column,** scattered over its whole area, interspersed with the long fibres of the posterior roots, but especially numerous at certain points, which are hence known as *endogenous tracts of the dorsal column.* These are—The ventral tract of the dorsal column ("cornu-commissural bundle"), in the neighbourhood of the commissure, and the medio-peripheral tract of the dorsal column ("septo-marginal bundle"), near the spinal periphery and the septum. The former is composed chiefly of ascending, the latter mainly of descending, intersegmental fibres.

III. Root Fibres.

These take their origin in the cells of the ventral horn, and pass by way of the anterior roots to the peripheral nervous system, where they are distributed to muscles, or connect with elements of the sympathetic system.

It will be advisable here to describe *the topography of the long spinal tracts,* as shown in transverse section. In Fig. 4 the ascending tracts we have so far dealt with are depicted on the left of the figure, the descending on the right.

Lateral Cortico-Spinal Tract.

The area of this tract, the "lateral pyramid area," is of greatest extent in the cervical cord, and diminishes steadily as one passes downwards. This is due to the fact that in each spinal segment *en route* a portion of the fibres

find their termination. In the upper portion of the cord the area has the form and position given in Fig. 4, in the dorsal portion of the lateral column. From about the third lumbar segment downwards, however, it is found at the dorsal periphery of the lateral column. Thus, at this lower level, it occupies the position which higher up belongs to the lateral cerebellar tract. The latter is absent from the caudal segments of the cord.

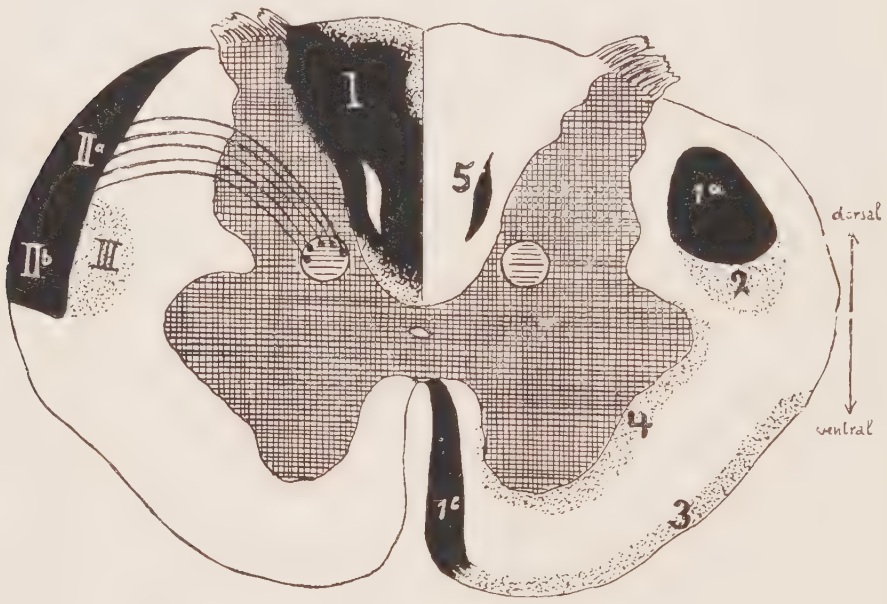


FIG. 4.—TOPOGRAPHY OF THE LONG SPINAL TRACTS.

Ascending (Centripetal) Tracts.

I = Long fibres of the posterior roots.
II = Spino-cerebellar tracts (*a*, dorsal; *b*, ventral = tract of Gowers).
III = Spino-thalamic tract.

Descending (Centrifugal) Tracts.

1 = Cortico-spinal tracts (*a*, lateral pyramid tract; *b*, anterior pyramid tract).
2–5 = Subcortico-spinal tracts (2, rubro-spinal and thalamo-spinal; 3, vestibulo-spinal; 4, tecto-spinal). 5, Descending fibres of the dorsal column.

Anterior Cortico-Spinal Tract.

The ventral pyramid area or antero-lateral descending tract borders the ventral median fissure on either side. This area diminishes so rapidly in passing down the cord that it is no longer discoverable in transverse sections of the lumbar and sacral regions.

Rubro-Spinal Tract.

This tract has a common area which forms a ventro-lateral continuation of that belonging to the lateral pyramid tract. It can be traced into the lumbar cord.

Tecto-Spinal Tract.

The fibres of this tract are found in the ventro-lateral column, in the zone bordering on the grey matter.

Vestibulo-Spinal Tract.

This forms a narrow fringe at the periphery of the ventro-lateral column.

Long Tracts of the Posterior Roots.

These occupy the area of the dorsal columns. At the positions which have been described as " endogenous areas " they are thinned out, being here more closely interspersed with intersegmental fibres. The descending fibres form the comma tract of Schultze.

Spino-Cerebellar Tracts.

These fringe the dorsal half of the lateral column. The dorsal portion of this field forms the *lateral cerebellar tract;* the ventral, *the tract of Gowers.* These tracts are not found at a lower level than the third lumbar segment.

Spino-Thalamic Tract.

This tract is situated in the lateral column, opposite the base of the dorsal horn. Where the tract of Gowers is present, the spino-thalamic tract is in close apposition to it medially.

In Fig. 4 those tracts which are constituted of compact bundles of fibres are represented in black. More scattered or interspersed tracts are figured by dotted areas. It must be borne in mind, however, that even in those tracts which consist mainly of closely associated fibres of the same class a certain number of intersegmental fibres are present (in the pyramid tracts, for instance, the ascending and descending *fibræ propriæ endopyramidales*). Areas also frequently overlap each other, so that, for instance, spino-thalamic and thalamo-spinal fibres are intermingled. In brief, there are in the spinal cord no perfectly homogeneous tracts.

II. CELLS AND CELL-GROUPS OF THE SPINAL CORD.

A distinction is to be made between "tract" cells and "root" cells (see Fig. 5).

(*a*) Tract Cells (Cellulæ Funiculares).

From these cells arise the tracts which we have enumerated on pp. 8 and 9, under the headings "Centripetal Fibres of the Second Order" and "Intersegmental Fibres." They lie scattered over the dorsal horn and a large part of the

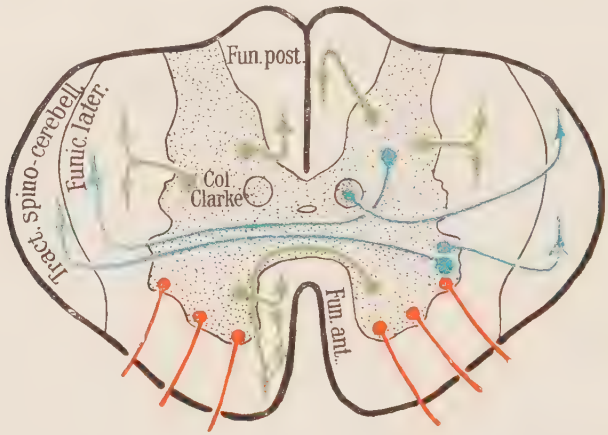


Fig. 5.—Endogenous Spinal Fibres and their Cells of Origin.

Red = Root cells.
Blue = Cells of secondary centripetal neurons } Tract cells.
Green = Cells of association fibres }

ventral horn, and are in part erratic, or without any definite grouping, in part united into cell complexes, of which the most important is the *column of Clarke*, at the base of the dorsal horn.

(*b*) Root Cells (Cellulæ Radiculares).

From these cells arise the anterior roots. They lie without exception in the ventral horn, and are characterized by their more or less definite grouping. In the lumbar and cervical regions, for instance, where the expansion of the grey matter is most considerable, one can distinguish an antero- and a postero-lateral, an antero- and postero-mesial, and finally a central group. Later we shall give more detailed consideration to these groups, which contain nuclei and nerve centres. The most important are the lateral groups, from which springs the chief contingent of the anterior root fibres.

All the cell-groups of the ventral horn are distinguished by their very copious blood-supply. While radial branches from the vaso-corona, the arterial "wreath" which forms the connection between the anterior and posterior spinal arteries, and surrounds the spinal periphery, supply the white substance and the greater part of the dorsal horns, a special arterial trunk penetrates into the ventral horn, on the right side or the left, according to the level. This

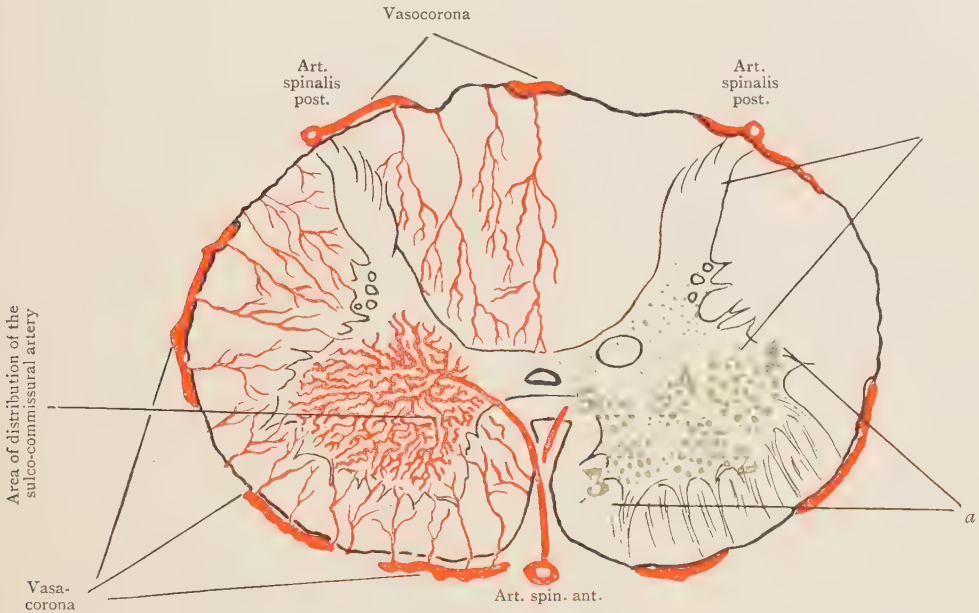


FIG. 6.—DISTRIBUTION OF BLOODVESSELS AND NERVE CELLS IN THE TRANSVERSE SECTION OF THE CORD.

a, Root cells: (1) Postero-lateral group; (2) antero-lateral group; (3) antero-mesial group; (4) central group; (5) postero-mesial group.

sulco-commissural artery forms, in the substance of the ventral horn, a rich and intricate network, which holds the cell-groups of the anterior roots enweaved in its meshes. Only in the most peripheral portion of the ventral horn, close to the boundary of the white matter, does the vaso-coronal system share in the nourishment of the grey matter. We shall see that the existence of two quite distinct arterial "territories" in one and the same transverse section of the cord is a fact of the greatest clinical significance.

CHAPTER II.

We now proceed to consider the physiology of the structures which in the foregoing chapter we have looked at from a purely anatomical point of view. We shall thus learn what functions the separate components of the spinal cord, its tracts and nuclei, subserve, and, conversely, shall be placed in a position to infer, from the presence of definite physio-pathological phenomena, lesions of definite areas in the cord.

The spinal cord is an organ of fourfold physiological significance, performing at once sensory, motor, vaso-motor, and trophic functions.

It will be as well to group its elements for consideration under the above four heads.

I. SENSORY APPARATUS OF THE SPINAL CORD.

Sensory impulses reaching our consciousness or subconsciousness, and due to occurrences within the sphere of trunk or limbs, make their way into the cord through the spinal ganglia and the posterior nerve roots. Corporeal sensibility in the broadest sense of the term is only normal, therefore, in the presence of a normal and intact system of posterior roots and connecting neurons. Corporeal sensibility is not a term of single, sharply defined meaning. In its analysis of the conception, physiology distinguishes between sensations of pressure, position, cold, warmth, pain, motion, and so on.

Clinical medicine does not go so far, but in its semeiological investigation of corporeal sensibility, confines itself, in the main, to the testing of four principal functions.

1. Tactile Sensibility.

This is tested by means of a pledget of cotton-wool, a camel-hair brush, the finger, etc. Its diminution is termed "tactile hypæsthesia," its abolition "tactile anæsthesia."

2. Temperature Sense.

In studying this we test the subject's distinguishing power for cold and warm objects. The conditions of imperfect or absent temperature sense are known respectively as "thermo-hypæsthesia" and "thermo-anæsthesia."

3. Sensibility to Pain.

Pricks with a needle, pinching a fold of skin, etc., will enable us to form conclusions here. Diminution=hypalgesia; abolition=analgesia.

The foregoing may be grouped together under the heading "Superficial Sensibility," the integument being in each case the seat of the stimulations

to be investigated. Superficial sensibility may, from another point of view, be separated into two subdivisions, a "protopathic" and an "epicritical." The former, which, during recovery from a cutaneous anæsthesia, is the first to be re-established, includes perception of painful superficial stimuli and the power to recognize wide differences of temperature; the latter is much slower to return, and is tested by light stroking, by estimating the power to recognize slight differences of temperature, and finally by observing the capacity to distinguish whether one or both points of a pair of compasses is applied to the surface (Weber's test). For further detail *vide infra* in the section on the brain. Pathological increase of tactile or temperature sensibility is described as "tactile" or "thermic hyperæsthesia," increased sensibility to pain is termed "hyperalgesia."

4. **Deep Sensibility.**

By this term we understand the sum of the centripetal impulses which reach our central nervous organs from the muscles, tendons, bones, joints, etc. A portion of these impulses crosses within the brain the threshold of our consciousness, and makes us aware of the position of our limbs, the amount of flexion or extension, abduction or adduction at particular joints, the extent of purposive movements, etc. Here also must be included the sensation of vibration ("pallæsthesia"), felt when a vibrating tuning-fork is placed on superficially situated parts of the bony skeleton. Another portion, however, does not reach the sensorium, but regulates subconsciously the motor mechanism which is called into play in the carrying out of all complicated and combined movements—*e.g.*, standing or walking. This portion provides for the preservation of equilibrium, the harmonious performance of the function of locomotion, the "synergetic" action of functionally related groups of muscles, and so on.

More or less marked deficiencies in the sphere of the deep sensibility serve to fill in the symptom pictures of ataxia, inco-ordination, asynergia, etc. As the sense of motion and of position possessed by our fingers plays the chief rôle in the recognition of objects by touch with the eyes shut, "astereognosis,"* or the inability to appreciate form by means of palpation, is also an expression of disturbed "deep sensibility." Inability to feel vibration is termed "pallanæsthesia."

The fibres which serve for the conduction of sensory impressions of the above four chief categories separate from each other immediately after their entrance into the cord.

1. Tactile sensations are conducted towards the brain by both long and short fibres of the posterior root system (*cf.* pp. 5-10, and Fig. 2)—some on the

* As the word "agnosis" is generally used to denote loss of some perception due to a defect or lesion in the psychical sphere, the abolition of the power of distinguishing form due to disturbance of the deep sensibility should be termed "stereo-anæsthesia" rather than "astereognosis."

same, some on the opposite side of the cord, the latter via the spino-thalamic tract.

2. Thermal sensations.

3. Sensations of pain are conducted exclusively by short fibres of the posterior root system to cells in the dorsal horn, and from thence by the above-described centripetal fibres of the second order, which ascend to the thalamus in the opposite lateral column.

4. The deep sensibility* makes use of two routes—(*a*) by long fibres of the posterior root system and the dorsal columns to the thalamus and brain; (*b*) by medium fibres of the posterior root system, the column of Clarke, and the lateral cerebellar tracts, to the cerebellum. Both routes ascend uncrossed. The cerebellar fibres serve for the conduction of subconscious tactile and co-ordinatory perceptions; the cerebral for the same purpose, and, in addition, for the conduction of conscious perceptions of position and motion to the cerebrum.

II. MOTOR APPARATUS OF THE SPINAL CORD.

By motility, in its broadest sense, we understand, not merely the phenomena of voluntary movement, but also those of tonus and reflex.

(*a*) Voluntary Motion.

Psycho-motor impulses arise, in greatly preponderating measure, from the motor centres of the cortex, and are conducted to the spinal centres in the ventral horns along the pyramid tracts. The cortico-spinal tracts are therefore to be regarded as the chief routes for motor impulses. They are, however, assisted in their task by a number of secondary tracts.

A lesion, therefore, putting the pyramid tracts completely out of action, may cause, not complete paralysis, but merely a serious diminution of motility, or paresis, the secondary tracts acting, up to a certain point, vicariously for the pyramidal. These secondary tracts are those which we have denominated subcortico-spinal tracts. Their centres of origin (the red nucleus, the tectum opticum, the nucleus of Deiters) have communications with the motor cortex, thanks to which, even in the presence of lesions of the pyramid tracts, some psycho-motor impulses succeed in reaching the cell-groups of the ventral horn by a circuitous route.

The centres for the whole muscular system are situated among the cell-groups of the ventral horns; thus each separate muscle is innervated from, and represented by, a particular ganglion-cell group in the cord. The exact principles governing localization in this connection will be considered in detail

* In the peripheral nervous system the fibres conveying impulses of the deep sensibility run in the nerves of the muscles, hence the deep sensibility is not impaired by lesions of the branches of the cutaneous nerves.

when the subject of regional diagnosis *qua* level, or segmental diagnosis (Höhendiagnostik), comes to be dealt with. Here we have merely to state clearly that destruction of the motor cells of the ventral horn leads to complete paralysis of the muscles governed by them.

(*b*) Tonus and Reflex.

A mechanical stimulus, such as striking or rubbing, acting on particular parts of the body (certain tendons, bones, special areas of skin or mucous membrane), gives rise, under normal circumstances, to correspondingly definite motor phenomena (contraction of particular muscles). We speak of these as reflexes. Apart from this, however, subconscious afferent impulses of less intensity are continuously passing from every part of the body, deep and superficial alike, to the central nervous organs, and these bring about, also reflexly, a moderate but continuous contraction of the whole muscular system. This is known as muscular tonus. It may be defined as "the particular degree of tension which gives to our muscles the capacity for prompt response, by a contraction, to a voluntary nervous impulse." That it is brought about reflexly is shown by the fact that section of the posterior roots—in other words, suppression of centripetal impulses—places the muscles in a condition of complete atony. That under normal conditions centripetal impulses are continuously at work, producing muscular tonus in a reflex manner, is proved by the fact that in healthy persons the condition of tonus never ceases to exist even in sleep. The muscular system of a sleeper is never so completely relaxed as that of a corpse, and it is only in the more advanced stages of narcosis that tonus is completely abolished.

It is plain from the foregoing that in the mechanism of spinal reflex and spinal tonus* we have both centripetal and centrifugal components, on whose integrity depends the normal bringing into play of these phenomena, so essential to the correct performance of voluntary movements (*cf.* Fig. 7).

The centripetal limb, so to speak, of the reflex and tonus arc lies, of course, in the posterior roots, the only structures through which centripetal tracts enter the cord. Equally, of course, the centrifugal limb is furnished by the anterior root system—*i.e.*, the peripheral motor neurons. The connection between the two is established by the terminal splitting of certain of the posterior root fibres among cells of the ventral horn.

These "reflex collaterals" belong to the short fibres of the posterior root system. In addition to this simplest form of connection, in which the whole

* We purposely defer consideration of another form of tonus, which may be called cerebellar contrasted with the spinal form, to the sections on the cerebellum and corpus striatum. That the sympathetic system also (*vide infra*, pp. 20 and 21) is concerned in the production of tonus in certain portions of the muscular parenchyma is maintained in recent physiological works. It is, however, so far without regional diagnostic significance.

reflex arc lies at one and the same spinal level, there are also more complicated forms in which there are further connecting neurons between the centripetal and centrifugal limbs, by which a stimulus may be conveyed from a posterior root to an anterior root of a higher level, or to several anterior roots—perhaps even, by a detour, to the corresponding anterior root, etc. These conditions, however, need not detain us longer at this stage of our inquiry.

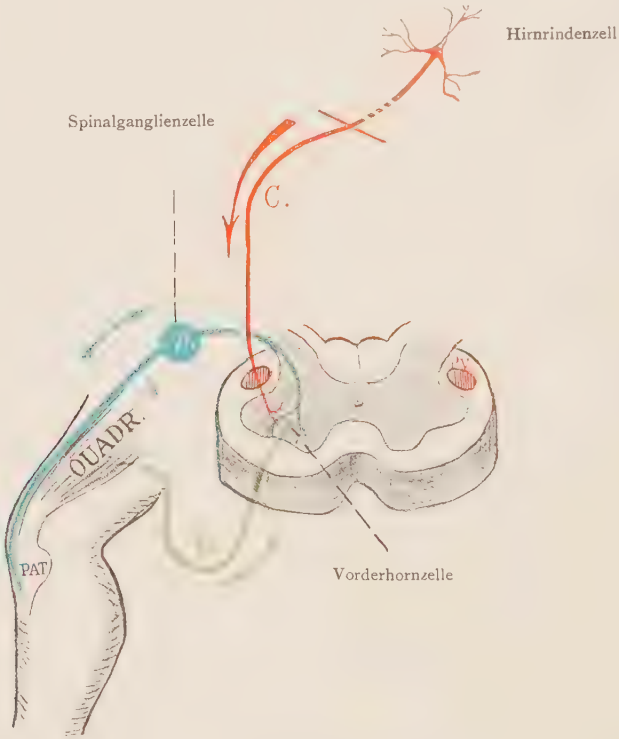


FIG. 7.—SCHEMATIC REPRESENTATION OF THE MECHANISM OF SPINAL REFLEX.

Blue: Afferent } Limb of reflex arc.
Green: Efferent }
Red: Cerebral inhibition of reflex.
Lesion of neuron A } Abolition or diminution of reflex and tonus.
,, ,, B }
,, ,, C = Hyperreflex and hypertonus.

We know, however, that certain cortico-spinal fibres also have their terminal ramification among the same motor cells, in whose neighbourhood the "reflex collaterals" of the posterior root system split up. These also have their part to perform in bringing tonus and reflex into play. They fulfil the function of a governor or regulator, acting inhibitorily, and never allowing reflex movements or tonus to be developed beyond a certain definite and serviceable degree. Without this inhibitory action of the pyramid tracts the phenomena of reflex

would not be so constant in character as they are. When those tracts are destroyed, tonus increases to such a degree as to be a serious hindrance to the performance of voluntary movements, and we find the slight concussions of the body produced in locomotion liberate reflexes sufficiently powerful to produce a markedly disturbing effect.

The following simple rules, therefore, may be laid down concerning tonus and reflex:

Lesions of the anterior or posterior root systems cause *diminution* or *abolition* of tonus and reflexes, lesions of the pyramid system cause *increase* of tonus and reflexes.

III. THE TROPHIC APPARATUS OF THE CORD.

The circumstance that each nerve cell constitutes the trophic centre for the nerve fibres arising from it—we recall here the *Wallerian law* mentioned on p. 4—has, as a consequence, the fact that the root cells of the cord govern the nutrition of that part of the peripheral nervous system which takes its origin from them—*i.e.*, the motor nerves throughout the body. In an analogous manner the integrity of the sensory nerves throughout the body is dependent on that of the spinal ganglia. The trophic influence of the cells of the ventral horn, however, is not confined to the peripheral motor nerve fibres. The terminal motor apparatus, the muscles themselves, are also subject to it. When the cells of the ventral horn are destroyed, the muscles innervated by them undergo an atrophy which is described as degenerative, to distinguish it from the simple atrophy of disuse. It is characterized, from the point of view of pathological anatomy, by a rapid disappearance of contractile elements, and their replacement by a similarly rapid growth of fat and fibrous tissue, and, clinically, by the reaction of degeneration.

Where this latter comes to its complete development, the galvanic and faradic irritability of the nerves is extinguished within about fourteen days. At the same time, the irritability of the muscles to direct faradic stimulation diminishes and disappears, while, on the other hand, their irritability to galvanic stimulation increases. The galvanic contraction, however, instead of being prompt and lightning-like, is slow and "creeping," and the contraction formula so altered, that the anodal closing contraction takes place with an equal or a weaker current than does the kathodal closing contraction. After about two months, however, the direct galvanic irritability of the muscles begins also to diminish, and after the lapse of a few more months is extinguished. For an account of partial and modified forms of the reaction of degeneration, works on electro-diagnosis must be consulted.

The cells of the ventral horn exert a trophic influence over the bony skeleton also When destroyed in growing subjects, a more or less marked inhibition

is noticed in the further growth of the bones in the region of the affected muscles. Abnormal brittleness of the bones may also result under like circumstances.

It would seem that the trophic influence of the cells of the ventral horn is to a certain extent called into action reflexly by elements of the posterior root system, inasmuch as trophic disturbances of the skeleton and muscular system are sometimes found to follow (though at a considerable interval) destruction of the posterior roots.

IV. THE SPINAL VASO-MOTOR MECHANISM.

Vaso-motility, as is well known, is under the direct governance of the sympathetic system. Experimental physiology teaches that vaso-constrictor fibres arise from the so-called "lateral" ganglion system or sympathetic chain of ganglia; while, on the other hand, vaso-dilator fibres arise from the "collateral" ganglion system—*i.e.*, from the other cell complexes included in the tracts of the sympathetic nerves. It is true that the existence of vaso-dilator fibres has only been experimentally proved in the case of certain special nerve trunks of the body and extremities—*e.g.*, the nervi erigentes and the sciatic. Vaso-constrictor fibres, on the contrary, are to be found generally distributed as components of the mixed spinal nerves. We know accurately how they reach the latter: it is by way of the grey *rami communicantes*, which pass from the separate ganglia of the sympathetic chain to join themselves to the corresponding somatic nerves.

While the term "sympathetic" is now generally reserved for the nervous elements of the ganglion chain system, the visceral nervous apparatus in the cranial and pelvic regions is spoken of as the "para-sympathetic" or "autonomous" system. The "cranial-autonomous" system connects with the mid-brain and the medulla oblongata, the "pelvic-autonomous" with the second, third, and fourth segments of the sacral cord. To the cranial-autonomous system belong the ciliary, otic, and spheno-palatine ganglia (*vide infra* in section Brain Stem); to the pelvic-autonomous the hypogastric plexus and the pelvic ganglia.

The ganglion cells, however, of the sympathetic chain, of which vaso-constrictor fibres are to be regarded as the axis cylinders, themselves constitute connecting neurons for centrifugal offshoots of certain spinal cells. The latter, the spinal centres for vaso-constriction, lie in the ventral horns, and, for the most part, in their mesial cell-groups. Their offshoots issue from the cord with the anterior roots, to pass along the white *rami communicantes* to the sympathetic chain. These spinal vaso-motor centres, however, are themselves governed by a bulbar vaso-motor centre, situated in the neighbourhood of the *upper olive*, whose stimulation is followed by a general vaso-constriction, while general loss of vascular tone follows its destruction. That, further, above this bulbar centre there must stand still a supreme centre, in the higher brain, is

shown (apart from infrequent and somewhat conflicting experimental work) by the fact that psychic emotions may have, as a consequence, blushing or pallor.

We have endeavoured to present in schema form (Fig. 8), and in accordance with the foregoing, the mechanism of vaso-motility, with its four neurons—cerebro-bulbar, bulbo-spinal, spino-sympathetic, and sympathetico-muscular.

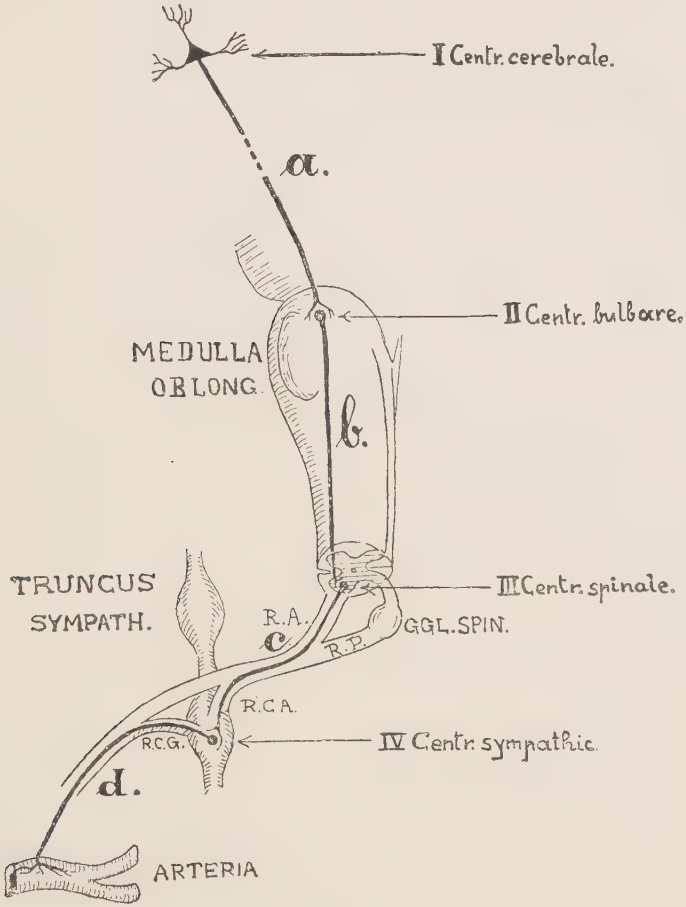


FIG. 8.—DIAGRAM OF THE MECHANISM OF VASO-MOTILITY.

a=Cerebro-bulbar vaso-motor tract; *b*=Bulbo-spinal vaso-motor tract; *c*=Spino-sympathetic vaso-motor tract; *d*=Sympathetico-muscular vaso-motor tract; I-IV=Primary and subordinate centres; R.A.=Radix anterior; R.P.=Radix posterior; R.C.A.=Ramus communicans albus; R.C.G.=Ramus communicans griseus.

Vaso-dilator fibres are not represented in the schema, as they are not of general occurrence, but are only found in certain special regions. As a matter of fact, they are practically ignored clinically. Broadly speaking, we regard loss of vascular tone as due to vaso-constrictor paralysis, and vaso-constriction as due to vaso-motor stimulation.

It is easy, then, to understand that lesion of those parts of the spinal grey matter in which lie spinal vaso-motor cells has, as a consequence, vaso-motor paralysis, and, further, that, under certain circumstances, disconnection of the lateral columns, by interruption of the bulbo-spinal connections, will have the same effect. What these particular circumstances are we cannot state with precision from an anatomico-pathological point of view, for we do not yet possess exact topographic data concerning the tracts of the lateral columns which are involved.

Spinal vaso-motor paralysis, whether produced by destruction of the vaso-motor centres in the ventral or lateral horn, or by interruption of vaso-motor fibres in the lateral columns, seldom reveals itself, and then only in its earliest stages, by redness and heat of the skin. A picture of an apparently opposite nature is more frequently presented, one that may supervene on and replace the other, but may also develop without any obvious stage of redness. The integument, in fact, in the regions innervated by the affected sections of the cord, becomes cold and cyanotic. The cyanosis is explained by the chronic obstruction to the capillary circulation which follows the removal of vascular tonus, the coldness (which may amount to a diminution in temperature of 10°) by the disturbance of cutaneous nutrition due to the cyanosis.

A counterpart of motor reflexes is presented by the vaso-motor reflexes, of which the so-called reflex erythema, named also *dermographia dolorosa* or *spinalis*, has clinical significance. If the skin—the phenomenon is best seen on the thorax—be irritated by drawing the point of a needle over it or by a simple prick, there appear around the irritated part and radiating from it tongue- or flame-shaped areas of reddening, occasionally also islets of vaso-dilatation surrounded by normally coloured skin. This reflex vaso-dilatation takes some time to disappear. It is abolished in destructive lesions of the part of the spinal cord under consideration, or of the sympathetic or peripheral nervous systems, and is to be distinguished from the so-called " dermographia peripherica " caused by stroking the skin with a blunt object, and characterized by white, rosy, or red stripes, presenting sometimes a raised, prominent, central portion, due to serous infiltration (urticaria factitia).

APPENDIX.

The mechanism of perspiration is very similar to that of vaso-motility. Here also we have cerebral, bulbar, spinal, and sympathetic centres. The schema of Fig. 8 will serve as a description of the nervous elements of the sweat mechanism, if we merely substitute a sweat gland for the bloodvessel. The spinal sweat centres seem, like the spinal vaso-motor centres, to be scattered throughout the cord, and to have their cells in the ventral horns in the neighbourhood of the motor ganglion cells and in the lateral cornua. Their injury or destruc-

tion is followed by diminution or abolition of the sweat secretion in the regions of the body involved—hyphidrosis or anidrosis. One would meet with these phenomena oftener than is at present the case were it the routine practice in diseases of the spinal cord to employ diaphoretics in the endeavour to detect abnormalities of the function.

CHAPTER III.

HAVING passed in review the various neuron systems of the spinal cord both from an anatomical and a physiological point of view, the clinical symptoms and symptom groups present in any given case should make it possible to answer the question—Which of those neuron systems is destroyed or damaged by a pathological process ?—*i.e.*, which parts of the transverse section of the cord are involved in the latter ?

Diseases and injuries of the cord announce their presence as a rule either—

(*a*) By disturbances of motility; or
(*b*) By disturbances of sensation; or
(*c*) By a mixture of both the foregoing.

Motor and sensory conditions are the only diagnostic guiding stars in the symptomatology of spinal diseases. Trophic and vaso-motor phenomena come so far behind them in significance, that, in questions of regional diagnosis, they are only brought in as secondary arguments and in special cases.

I. THE REGIONAL DIAGNOSTIC SIGNIFICANCE OF MOTOR DISTURBANCES.

Paralysis or paresis follows, as we have seen, interruptions of continuity, either in the cortico-spinal or in the spino-muscular neurons.

The former occur in the great majority of cases in the lateral columns; the latter have their seat exclusively in the ventral horns and anterior roots. When, in spinal affections, therefore, we meet with paralysis or paresis, the first question we have to decide is this: Is it a lateral column affection, or one of the ventral horn and anterior roots ? In some cases, it is true, we have to consider the possibility of a combination of the two conditions.

(*a*) Characteristic Features of Paralytic Affections due to Lesions of the Lateral Columns.

(*a*) In the first place, the paralysis is here not complete, but is rather of the nature of paresis; for the anterior pyramid tracts remain, and also the subcortico-spinal tracts, which carry motor impulses from the brain, and inner-

vate the motor centres in the ventral horn by a circuitous route. Where the affection is one of the lateral pyramid area alone, all the subcortico-spinal tracts enumerated on pp. 4 and 5 come into play vicariously; where, however, the whole lateral column is affected, the anterior pyramid tracts may be assisted by a portion of the tecto- and vestibulo-spinal fibres.

(β) The paresis affects in about equal degree all the muscles of the affected extremity. In a given transverse section of the lateral pyramid area there are cortico-spinal fibres lying in close contact with one another, whose terminal ramifications are distributed over a wide stretch of the motor-cell columns of the ventral horns.

(γ) Rigidity and spastic stiffness of the paretic muscles is a noticeable feature, a consequence of the cessation of tonus-inhibiting impulses from the cortex, which, as was shown on p. 18, pass continuously along the pyramid tracts to the cells of the ventral horn. It is easy to satisfy oneself of the presence of this hypertonus by palpation, testing the resistance to passive movements, etc. Excitability to mechanical stimuli is also increased in the paretic muscles, owing to the presence of hypertonus.

(δ) There is an exaggeration of tendon and bone reflexes in the sphere of the paretic muscles, which is analogous to their general spastic condition, and susceptible of an analogous explanation (*cf.* p. 18, Fig. 7). With this exaggeration of reflexes, the phenomenon known as *clonus* may often be demonstrated in the affected musculo-tendinous mechanism. The phenomenon consists of a series of muscular contractions rapidly following each other in the affected muscles, and produced by a suddenly applied passive stretching of their tendons. By far the most frequently elicited is the ankle clonus (Achilles tendon); patellar clonus is not uncommon; wrist, finger, and elbow clonus are rare.

The condition of the skin reflex is far more variable than that of the tendon reflex. Exaggeration of skin reflex can only be demonstrated in a certain proportion of the cases, in which, owing to a pyramid lesion, the normal cortical inhibition is suppressed. Occasionally, in contrast to the exaggerated tendon reflex, the skin reflex is actually diminished or absent. It will be sufficient briefly to state the fact here; in the section on segmental diagnosis opportunity will offer to deal with this peculiarity more fully.

(ε) Infallible signs of a lesion of the pyramid tracts are a number of pathological reflexes, of which the most important is that known as *Babinski's* sign. It is characterized by a slow tonic hyperextension of the great toe elicited by irritation of the sole of the foot (*vide* Fig. 9). This constant and invariable sign may be associated with other motor phenomena affecting the lower limbs—*e.g.*, with a tonic or clonic plantar flexion or a tonic hyperextension of the other toes, or, further, with a slow spreading out of all the toes (*signe de l'éventail*) with simultaneous twitchings of the tensor fasciæ latæ, the adductors of

the thigh or the quadriceps, or, again, with a contraction, lasting sometimes for seconds, of the flexor muscles of the thigh and leg with the tibialis anticus ("flight" or "defence" reflex). Besides the "classic" method for eliciting the Babinski reflex—viz., irritation of the sole of the foot—there are numerous modifications which show that the "reflex eliciting" zone for a slow hyperextension of the great toe is not confined to the plantar surface. In the best-known variant, known as the reflex of Oppenheim, the same hyperextension is brought about by a vigorous stroking of the medial surface of the calf. Pricking the dorsum of the foot with a needle, kneading the gastrocnemius or repeated tapping over the metatarso-phalangeal region close to the middle of the *extensor hallucis longus* tendon may also be employed as stimuli. Other pathological reflexes which may supplement or replace that of Babinski are the *Mendel-Bechterew* sign, a plantar flexion of the toes caused by percussion applied to the side of the dorsum pedis, the reflex of Rossolimo, flexion and abduction caused by quick, sharp blows on the ball of the toes, Bing's reflex (paradoxal ankle reflex), a plantar flexion of the foot caused by percussion on the line between the two malleoli on the extensor aspect. As a pathological reflex in the upper extremity may be mentioned the modified radius reflex in which percussion over the distal end of the radius causes contraction of the digital flexors, the same stimulus causing, under normal conditions, flexion of the forearm at the elbow. These "pathological reflexes" are normally present in the infant during the first few months of life, and this is probably due to the fact that it is only after birth, during the early post-embryonal period, that the pyramids become fully effective in function through the clothing of their axis cylinders in a medullary sheath. No satisfactory explanation of these phenomena has yet been given. We can only suppose that the reflex stimulus, when not controlled from above—*i.e.*, by the cerebral centres—forces its way into tracts which are normally closed to it.

FIG. 9.—BABINSKI'S SIGN.

(ζ) The same may be the case when a voluntary impulse rather than a reflex stimulus is in question, so that a particular intended movement of the paretic muscles is accompanied by unintended movements. Thus it may be

impossible to flex the lower limb on the trunk without bringing about simultaneously a vigorous dorsal flexion of the foot (*tibialis phenomenon or Strümpell's sign*). Involuntary hyperextension of the great toe may take place under the same circumstances (*toe phenomenon*). In the hand we have the *rädialis phenomenon* (involuntary dorsal flexion of the hand accompanying volar flexion of the finger); involuntary pronation may accompany flexion of the forearm (*pronation phenomenon*), and so on.

(η) There is no atrophy of the affected muscles—none, at least, except the late and never very marked "atrophy of disuse"—for the muscles remain, in spite of interruption of the pyramid tracts, in unbroken connection with their trophic centres, the cells of the ventral horn. For the same reason the muscle retains, with its anatomical integrity, its electrical excitability.

(θ) The signs of vaso-motor paralysis mentioned on p. 22, coldness and cyanosis, may be present in the parts affected by cortico-spinal paralysis. In such case the vaso-motor neuron in the lateral columns is probably interrupted. This is, however, not by any means always the case, nor are vaso-motor phenomena as a rule present in any marked degree.

The whole picture of paralysis due to affection of the lateral columns—*i.e.*, to interruption of the cortico-spinal tracts—may be described as a spastic symptom complex.

(*b*) Characteristics of Paralysis due to Affections of the Ventral Horn.

(α) The paralysis of the affected muscles is at first complete. No other structures can take on vicariously the work of injured motor neurons of the second order. As time goes on, there may be restoration of function to a certain degree (if, for instance, some of the originally merely damaged motor cells succeed in restoring themselves); this restoration is, however, always incomplete and always slow.

(β) It is seldom that all the muscles of an extremity are paralyzed as the result of a ventral horn lesion. This fact might be inferred from the anatomical conditions. We have seen above that in a transverse section the nuclei for the separate muscles lie separated from one another, scattered over an area of some extent, and are thus far less liable to be destroyed simultaneously by one and the same lesion than the closely compacted fibres, covering only a small area, of the pyramid bundle. Our discussion of *segmental* regional diagnosis will show us, further, that in longitudinal sections of the ventral horns this peculiarity is still more perfectly displayed, so that the nuclei of separate muscles of the same limb may be centimetres apart from each other. Lesions of this extent, however, are rare in spinal pathology.

(γ) The muscles affected are atonic (interruption of the centrifugal limb of the tonus-reflex arc). Absence of resistance to passive movements, looseness

of articulations owing to deprivation of muscular support, possibility of enormous hyperextension, "professional contortionist" positions, marionette attitudes, betray this atony with sufficient clearness. With this complete atony is associated suppression of direct mechanical excitability.

(δ) The reflexes disappear (interruption of the centrifugal limb of the reflex arc).

(ε) There are no involuntary associated movements.

(ζ) Lesions of the ventral horn centres are very rapidly followed by severe atrophy of the muscles in trophic dependence on them. This degenerative atrophy is accompanied by the "reaction of degeneration."

(η) Vaso-motor disturbances are much more severe, and appear much more frequently than is the case in cortico-spinal paralysis. The important vaso-constrictor centres are especially liable to come within the range of the lesion. In addition to marked coldness and cyanosis of the integument, lowering of blood-pressure (an evidence of loss of tone in the arterial trunks) is also to be noticed.

The fact that the ventral horns, whose situation is a much more protected one than that of the pyramid tracts, and whose position in a transverse section is not far removed from that of the latter, should so often be the seat of pathological processes, which those tracts escape, and thus give rise to paralysis of purely peripheral (spino-muscular) type, is to be explained by reference to the conditions of spinal blood-supply described on p. 13. Owing to their separate vascular supply, the anterior cornua are, nosologically, to a large extent independent of the remaining structures in the spinal transverse section. Owing to the very richness of their vascularization, they are specially liable to injury from noxious matters reaching them by way of the blood-stream, and to morbid processes of a periarteritic nature—*e.g.*, poliomyelitis anterior. On the other hand, the rich anastomosis of the sulco-commissural artery protects the ventral horn as a rule from the ischæmic degeneration which, in the angio-sclerotic processes of syphilitic endarteritis, for example, is so rapidly destructive to the more sparingly arterialized lateral columns. This selection for isolated attack, either of the central or the peripheral motor neurons, is also often seen in systemic degenerations; both may, it is true, be attacked simultaneously, as the condition known as amyotrophic lateral sclerosis sufficiently proves—indeed, such simultaneous lesions of protoneurons and deuteroneurons come under observation occasionally from the most diverse causes.

(*c*) Paralytic Phenomena due to Combined Lesions of Lateral Columns and Ventral Horns.

That this "combined" form of paralysis "resembles" the cortico-spinal type, in so far as the extent of the paralyzed region is concerned, is easy to understand; equally natural is it that otherwise, and on the whole, the features

of spino-muscular paralysis should dominate the clinical picture. The paralysis of most of the affected muscles is complete, and atrophy and the reaction of degeneration set in. Up to the point, however, at which atrophy brings about the destruction of the muscle, and the reaction of degeneration gives place to a complete absence of reaction, phenomena of hyperreflex and hypertonus may betray the lateral column degeneration lurking, so to speak, behind the degeneration of the anterior horn. During these comparatively early stages the reflexes are exaggerated, as also is the direct mechanical excitability. Further—where the paralysis affects the foot—hyperextension phenomena, Babinski's and Oppenheim's signs, may be elicited for a time, as also the Mendel-Bechterew phenomenon (dorsal-pedal reflex).

Spinal Paralyses.

(*a*) *Lateral Column Type.*	(*b*) *Ventral Horn Type.*	(*c*) *Combined Type.*
Paresis	Paralysis predonderating	Paralysis preponderating
General distribution	Distribution to individual muscles	Distribution general
Hypertonus	Atony	Indications of hypertonus, later atony
Hyperreflex	Absence of reflex	Hyperreflex, giving way later to absence of reflex
Associated movements frequent	No associated movements	No associated movements
Hyperextension phenomena and dorsal-pedal reflex	No hyperextension phenomena nor dorsal-pedal reflex	Occasionally hyperextension phenomena and dorsal-pedal reflex, disappearing later
No degenerative muscular atrophy	Degenerative muscular atrophy	Degenerative muscular atrophy
No reaction of degeneration	Reaction of degeneration	Reaction of degeneration

Before we turn to a consideration of sensory symptoms, brief mention must be made of certain motor "irritation symptoms" (hyperkinesis) which point to the presence of morbid conditions which, without leading to complete destruction either in the cortico-spinal or the spino-muscular neuron complexes, are sufficient to bring about abnormal dynamic phenomena.

1. The so-called "spinal epilepsy" of Brown-Séquard. This by no means common phenomenon is the motor "irritation symptom" of lesions of the lateral column. It is a spontaneous clonus of the paretic extremities, coming on in definite attacks, and causing a more or less violent shaking of the extremities by alternate contraction of their flexors and extensors.

2. The well-known "fibrillary twitchings" which occur in functional neurotic states, and can be brought on even in normal subjects, by the application of cold, for instance, are a frequently occurring motor irritation symptom of lesions of the ventral horn.

II. THE REGIONAL DIAGNOSTIC SIGNIFICANCE OF SENSORY DISTURBANCES.

It will be advisable here to refer to Fig. 2, and to recall, in connection therewith, what was said on pp. 15 and 16 concerning the separate sensory tracts. It was pointed out that tactile impressions pass to the brain by both the routes L and K2; that the latter, exclusively, serves for painful impressions and for the temperature sense; while, finally, impressions of the deep sensibility are conveyed to our consciousness or subconsciousness partly by route L to the brain, and partly by route M to the cerebellum.

These facts being kept in mind, we may proceed without further introduction to make the statements here following:

1. A lesion of the posterior roots abrogates or diminishes sensibility in the region from which those roots receive centripetal fibres. Disturbances of the sensibility of the integument to tactile, painful, and thermal impressions are combined with ataxic and astereognostic (*vide* p. 15) phenomena; expressions of impaired deep sensibility.*

2. Lesions of the grey matter interrupt the routes for painful and thermal impressions, which, one and all, pass through it. Lesions, also, which interrupt the spino-thalamic tracts in the lateral columns may be followed by analgesia and thermo-anæsthesia, or by diminution of those respective functions (hypalgesia, thermo-hypæsthesia). Whether in the two above-mentioned conditions the tactile sense, which, as we have seen, makes use partly of the same routes, is affected or not, depends on individual factors—*i.e.*, on the compensatory powers of the posterior columns in the affected subject. As a rule it remains intact. This condition, in which sensibility to pain and the temperature sense are abolished, while the other sensory powers remain unimpaired, is known as "dissociated anæsthesia." When fully and typically developed, it is pathognomonic of central affections of the cord (hæmatomyelia, central gliosis, syringomyelia, certain intramedullary tumours, etc.).

3. If, however, the tactile sense is entirely abolished, both its routes, alike that through the posterior column and that through the lateral column, must be interrupted, save in the cases of posterior root lesion mentioned above. In a lesion of the posterior column alone tactile sensibility is impaired, but not abolished. If, in the upper portion of the cord, the lesion affects only the column of Goll, the impairment of tactile sensibility is confined to the lower half of the body (*cf.* Fig. 3 and p. 8).

4. Ataxia points (apart from the above-mentioned root lesions) to lesions either of the posterior columns or of the spino-cerebellar tracts. As a rule the

* Sensibility to vibration reacts with great delicacy to lesions of the centripetal tracts. It may be diminished, or even completely abolished, when all other symptoms of involvement of the posterior roots are absent—*e.g.*, in cases of neural progressive muscular atrophy.

clinical features of the case enable us to distinguish between posterior column ataxia and cerebellar ataxia. To give here a description of these two conditions would take us too far; they are, besides, fully described in every text-book. Posterior column ataxia, the ataxia of tabes dorsalis, is a familiar clinical picture to every medical man. We will confine ourselves here to enumerating the differential physio-pathological criteria. In cerebellar ataxia single movements of the extremities are fairly well performed—with much greater accuracy, at any rate, than is the case in posterior column ataxia (tabes). On the other hand, the power of co-ordinating these single movements, the power of combined movement, or synergia, is very gravely interfered with. Such combined movements play a most important part in the performance of the function of locomotion, in the maintenance of the erect position, etc. Hence the zigzag progression of the cerebellar ataxic, the swaying of his body when attempting to stand upright, etc. That a mixed lesion of the posterior columns and of the spinal cerebellar tracts causes a particularly severe ataxia of mixed character is shown by a study of Friedreich's disease.

Sensory "Irritation Symptoms."

Morbid processes affecting the system of the posterior roots at their entry into the cord or during the further passage of their fibres brainwards may, before they bring about the destruction of the structures attacked, give rise to severe sensory disturbances. A characteristic feature of these sensory phenomena is that they are referred, not to the cord itself, but to the periphery—*i.e.*, to that region of the integument, musculature or periosteum in which are situated the sensory end-organs of the "irritated" tracts. Patients are better able to describe with accuracy the course of these tearing and cutting pains when only single roots or single pairs of roots are affected—as, for instance, in the case of circumscribed new growths or tuberculous foci in the spinal column, in pachymeningitis cervicalis hypertrophica, etc. The lightning pains of tabes dorsalis belong also to this category. In the case of hæmorrhagic or traumatic foci in the substance of the cord, the localization of the pains is less defined. In such cases they affect, as a rule, considerable areas of the body, as there is a simultaneous irritation of many closely apposed fibres which form the continuation within the cord of numerous pairs of posterior roots. Occasionally, but very rarely, "root pains" may be referred to parts of the integument which, tested objectively, prove to be anæsthetic. This is the case where posterior roots have been recently severed, and where, consequently, the conduction of impulses from the periphery to the centres is interrupted; while from the central ends of the torn roots impulses of an irritative nature may, for a time (until Wallerian degeneration sets in), reach the sensorium (anæsthesia dolorosa). The same may also occur in recent lesions of the sensory tracts in the cord itself.

The paræsthesias and hyperæsthesias are also to be regarded as sensory irritation symptoms, corresponding, however, to less extensive impairment of nerve function. Under the former heading we have to deal with sensations of an abnormal character arising spontaneously (formication, pricking, burning, etc.); in the latter case we have excessive subjective reactions to ordinary applied stimuli—a light touch or gentle stroking, for instance, may give rise to sensations of severe pain.

When root pains accompany herpes zoster, we may locate the lesion in the spinal ganglion. The physio-pathological basis of the vesicular eruption is at present obscure; but, bearing in mind the mechanism affected, it would seem possible that connections with the sympathetic play some part in the matter.

III. REGIONAL-DIAGNOSTIC SIGNIFICANCE OF MIXED SENSORY AND MOTOR DISTURBANCES.

The most important and typical forms of such mixed affection that we meet with in spinal pathology are—

1. Simultaneous affection of the lateral and posterior columns.
2. Disease or injury affecting the cord in transverse section. The lesion may be partial or complete.
3. Lesions interrupting the nerve channels of one lateral half of the cord (Halbseitenläsion).

(*a*) Simultaneous Affection of the Lateral and Posterior Columns.

This type is not very rare, and may be produced in various ways. In the first place, true systemic degenerations may affect simultaneously the posterior columns and the pyramid and lateral cerebellar tracts. Again, a chronic meningitis, especially one of syphilitic origin, may be complicated by a myelitis whose tendency is to pass inwards from the periphery, and involve by preference the posterior and postero-lateral columns. Thirdly, affections of the arteries, of sclerotic or luetic-endarteritic type, lead not infrequently to an ischæmic tissue necrosis, which, spreading inwards in wedgelike form from the periphery —a consequence of the anatomical distribution of the branches of the vaso-corona (*vide* Fig. 6)—attacks and destroys by preference the posterior columns and the lateral pyramid and lateral cerebellar tracts.

In these cases, then, centripetal channels and the most important centrifugal nerve-fibre complex are, owing to anatomical association with one another, simultaneously attacked. It is easy, therefore, to understand that the clinical picture likely to be presented is one combining ataxic and hypæsthetic phenomena with muscular paresis. As we have seen, however, lesions of the centripetal system are followed by hypotonus and hyporeflex; while, on the contrary,

suppression of the cortico-spinal inhibition brings in its train exaggeration of tonus and reflex. In mixed lesions such as we are considering, therefore, influences of diametrically opposite tendency are at work, and the result will depend on the greater or less intensity of the morbid process in the different structures affected.

If the pyramid lesion governs the picture, we get the symptom complex:

Spastic-Ataxic Paraplegia, with Disturbances of Sensibility.

If, on the other hand, the pyramid lesion is less important than that of the sensory tracts, the picture presented may be described thus:

Ataxia and Absence of Reflexes with Motor Impairment.

In cases of the latter kind the pathological reflexes pathognomonic of pyramid lesions (Babinski's and Oppenheim's signs, Rossolimo's and the Mendel-Bechterew phenomenon) may be very marked. The so-called "associated muscular movements" may also be present, and thus clear evidence be given that the posterior column lesion is accompanied by an affection of the lateral columns.

(*b*) Transverse Division (Disease or Injury affecting the Cord in Transverse Section).

Transverse division of the cord, a condition brought about most frequently by fractures or dislocations of the vertebral column, Pott's disease, etc., much more rarely by a transverse myelitis, gives rise, when complete, to no difficulties of diagnosis. In this case we have complete abolition of sensation and of motion. The former extends to every sensory function, as all the centripetal tracts are put out of action. The latter is also absolute, not paresis, but paralysis, for there is interruption, not only of the pyramid tracts, but of each and every connecting route between the organ of the will and the spinal motor centres.

It is clear that sensibility will be abolished in those integumental areas whose nerves enter the spine at or below the site of the lesion (*vide* Fig. 10), and, similarly, that the paralysis will affect every muscle innervated from nerve cells of the ventral horn at the level of the lesion or on its caudal side. Further and more detailed data concerning this distribution will be given when we come to the study of segmental diagnosis. Here we need only make the preliminary statement that in a complete transverse lesion of the cord in the upper cervical region the simultaneous paralysis and anæsthesia affect the four extremities and the trunk (tetraplegic type); if the dorsal cord is divided, the lower extremities and the lower half of the trunk are affected; while if the lesion is in the lumbar cord, only the lower limbs are affected (paraplegic type).

As regards the character of the paralysis, we should expect it to take a spastic form (pp. 24-26); for below the seat of the lesion the reflex arc is intact, and, owing to the lesion, it is freed from cortico-spinal inhibition. In complete division of the cord in its upper part, however, we find in almost every case, at any rate in the early stage, complete muscular atony and abolition of reflexes in the paralyzed regions (*Bastian's* law). An attempt has been made to explain this as an effect of shock or as "diaschisis" (*vide* section on the Cerebellum). The explanation is not satisfactory, however, for in the first place the atony and absence of reflexes persist in many cases, and in the second place they may be found in non-traumatic cases (myelitis transversa). We will not enter here upon a detailed consideration of the various theories that have been put forward to account for the paradox. None of them admits of strict proof. Two bear examination better than the rest. According to the first, in division of the pyramid tracts, it is necessary for the development, not merely of the spastic condition, but of tonus and reflex also, that the other centrifugal exogenous tracts should be intact. The second theory would place the responsibility for grave interference with the functions of the deeper-lying posterior roots and ventral horn cells on the disturbances of the lymph and blood circulations which accompany a total transverse lesion. Personally, I incline to the latter of the two theories.

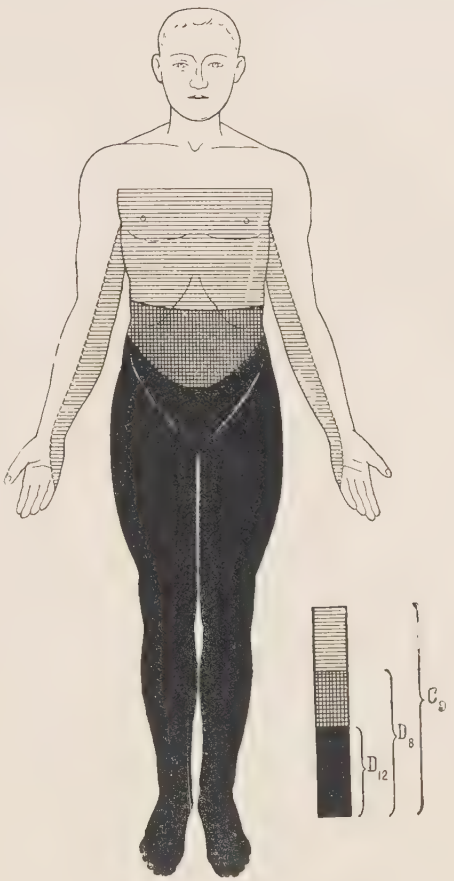


FIG. 10.—EXTENSION OF ANÆSTHESIA IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE SPINAL CORD.

In transverse lesions of the cord, not only the skeletal muscles, but also those of the bladder and rectum, are affected. The subject of sphincter innervation will be dealt with in the section on Segmental Diagnosis, where it may most appropriately be considered under the head of affections of the sacral cord. Here we need only state that in transverse division of the cord, wherever its seat, the bladder and the rectum are withdrawn from the influence of the will. Fæcal retention sets in. As regards the bladder, we have at first retention of urine. When, however, the distension of the bladder reaches a

certain point, it may empty itself involuntarily by reflex action (incontinentia intermittens).

We saw above (p. 22) that there are vaso-motor fibres in the lateral columns. This explains the vascular dilatation, due to vaso-motor paralysis, which one meets with in recent cases of complete transverse division, in the paralyzed region. Later, the condition (cold and cyanosis), described on p. 22 as marking chronic vascular relaxation, may supervene. The engorgement of the corpora cavernosa which is noticed in these cases is also due to vaso-motor disturbance, as, likewise, the pronounced tendency to the rapid development and extension of bedsores.

It is of very great importance, from the point of view of prognosis, to distinguish partial from complete transverse lesions.

The following distinguishing features should be kept in mind:

1. In partial transverse lesions the paralysis is as a rule not symmetrical, as in complete division; and, further, in contrast with the absolute incurability of the latter condition, there is a tendency during the first few weeks towards some measure of recovery. The identity of distribution, also, of motor and sensory disturbances which is noticed in cases of complete division is here generally absent, the motor disturbances affecting, as a rule, a wider area.

2. Vaso-motor and sphincter abnormalities are present only in slight degree.

3. Even when the lesion is situated at a high level, the patellar reflexes are never permanently abolished. As a rule they are exaggerated. A difference can often be noticed between the two sides.

On the distal side of the lesion, sensory, and sometimes also motor, irritation symptoms assert themselves (pains, anæsthesia dolorosa, paræsthesiæ, hyperæsthesia, epilepsia spinalis, etc.). In complete division, on the other hand, irritation symptoms are never present. As an irritation symptom one must regard also the marked priapism, which is to be distinguished from the simple engorgement of the corpora cavernosa present in cases of complete division. These matters also will be dealt with more fully in the section on Segmental Regional Diagnosis.

(*c*) **Unilateral Transverse Lesions (Halbseitenlasion).**

The phenomena which follow unilateral transverse division of the cord by trauma or disease are known as "Brown-Séquard's symptom complex." The term comprises the following morbid phenomena, which show themselves in those parts of the body distal to the site of the lesion.

(α) *On the Same Side as the Lesion.*

1. Motor paralysis.
2. Vaso-motor paralysis.
3. Disturbances of "deep sensibility."
4. Hyperæsthesia for tactile impressions.

(β) *On the Opposite Side.*

5. Disturbance of superficial sensibility—*e.g.*, to painful and thermal impressions.

We will consider the separate sections of the above syndrome in their order, with a view to a physio-pathological explanation.

1. Motor Paralysis of the Same Side as the Lesion.

This affects the regions innervated from ventral horn cells distal to the site of the lesion. Its development is easy to understand. The central motor tracts, especially the lateral pyramid tracts, convey to the spinal centres of the same side voluntary motor impulses. The paralysis has naturally a cortico-spinal—*i.e.*, spastic—character. In the isolated cases in which, immediately after the hemisection, the reflexes are weakened or abolished, this is only a temporary phenomenon, either due to shock or admitting of the explanations suggested on p. 33. Babinski's and Oppenheim's signs, Rossolimo's and the Mendel-Bechterew phenomenon can generally be demonstrated.

2. Vaso-Motor Paralysis of the Same Side.

The skin is, in the early stages, reddened and warmer than that of the opposite side. Later on this symptom simply disappears, or a chronic condition of cold and cyanosis takes its place. Physio-pathological explanation: Interruption of the vaso-constrictor fibres of the lateral column of the same side (*cf.* p. 22).

3. Disturbance of Deep Sensibility on the Side of the Lesion.

That the sense of position is seriously interfered with, or even entirely abolished, so far as the parts distal to and on the same side as the lesion are concerned, and that even when motility is restored to those parts, ataxia supervenes, can be readily understood if one bears in mind the fact that impulses of the deep sensibility are conveyed upwards almost exclusively on the same side of the cord, in the posterior columns and spino-cerebellar tracts.

4. Superficial Hyperæsthesia on the Same Side as the Lesion.

The explanation of this phenomenon is much less clearly established, and many are the hypotheses that have been put forward to account for it. Of these the most probable seems the following: As tactile impressions normally pass upwards, partly crossed (lateral columns), and partly uncrossed (posterior columns), only the crossed fibres can, after hemisection, convey impressions from the same side as the lesion. These fibres have thus extra work thrown upon them, and the cells of the dorsal horn, from which springs the crossed spino-thalamic tract, pass on to the latter "summated" stimuli, which at

first—*i.e.*, until the organism has accustomed itself to the new condition (this form of hyperæsthesia is a transitory symptom)—are felt as painful impressions

5. Superficial Anæsthesia on the Side Opposite to the Lesion.

This always affects sensibility to painful and thermal impressions, which is easily intelligible in view of their exclusively "crossed" conduction. In typical cases tactile sensibility is also, though less markedly, affected. This

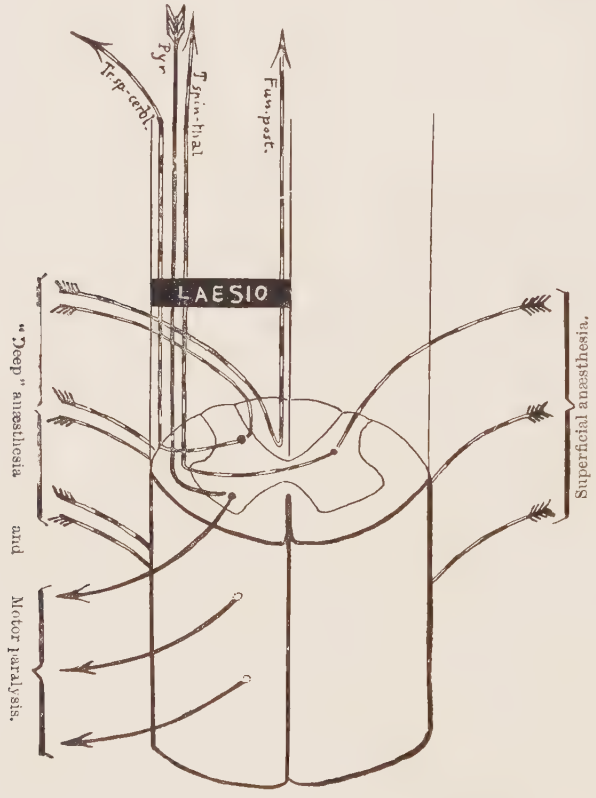


Fig. 11.—Brown-Séquard's Symptom Complex.

also is explained by the anatomico-physiological conditions. Tactile impressions are conducted to the brain partly, as we have seen, crossed (lateral columns), and partly uncrossed (posterior columns). In some cases, however, no tactile anæsthesia or hypæsthesia can be demonstrated. In these cases we must conclude that the uncrossed fibres in the posterior columns succeed in taking upon themselves, with extraordinary rapidity and completeness, the work of their affected coadjutors in the crossed lateral columns.

In the majority of cases lesions such as we are considering are situated in the dorsal region, and the condition produced is spoken of as *hemiparaplegia*. Fig. 12 is a diagrammatic representation of the symptomatology of such a case. The narrow zone of anæsthesia which is to be seen in the figure immediately

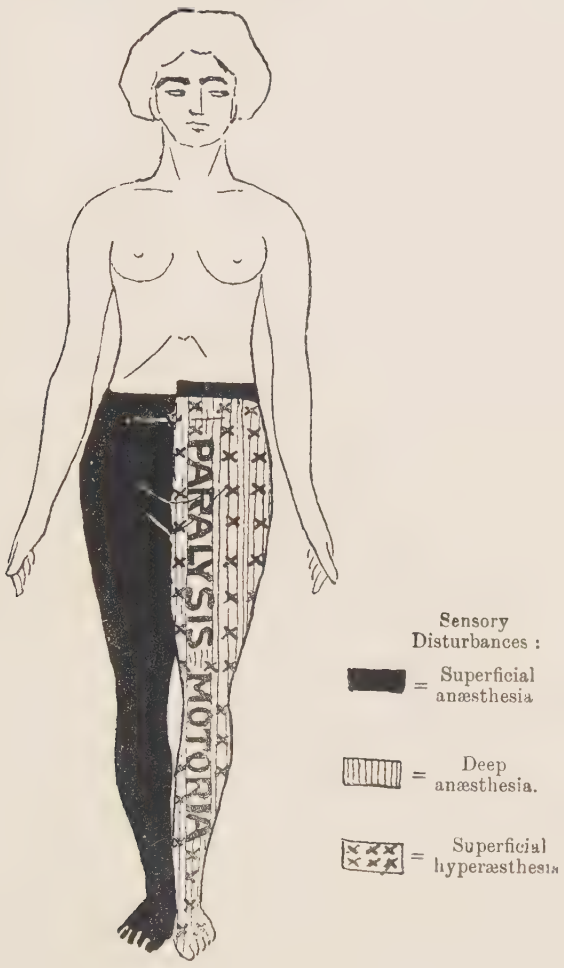


FIG. 12.—BROWN-SÉQUARD'S SYMPTOM COMPLEX IN LEFT UNILATERAL TRANSVERSE LESIONS OF THE CORD.

above the paralyzed region is due to the suppression of impulses from those posterior roots which enter the cord at the site of the lesion.

If the lesion is in the cervical region, the symptom complex is described as *spinal hemiplegia*. *Mutatis mutandis*, we have here the same distribution of motor and sensory disturbances. In lesions of the lower lumbar and sacral

cord this is not so. In these regions the separate segments are so closely pressed together that a lesion involving one lateral half of the cord allows few sensory channels to pass beneath it to the opposite side of the organ. Superficial anæsthesia is therefore generally present on the injured and paralyzed as well as on the uninjured side (*vide* Fig. 13). In very rare cases a pathological process may lead to a unilateral irritation instead of a unilateral abolition or diminution of function in the spinal cord. In these cases spastic phenomena are observed on the side of the lesion, and painful disturbances of sensation on the opposite side (" spasmodynia cruciata ").

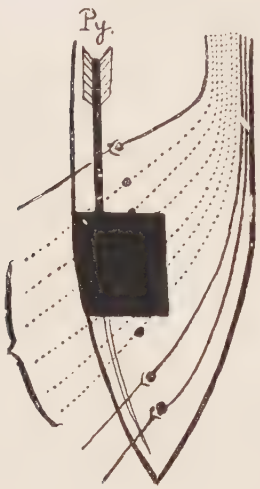


FIG. 13.—DIAGRAM TO EXPLAIN THE EXTENDED HOMOLATERAL ANÆSTHESIA FOUND IN HEMISECTION OF THE CORD IN THE LOWER LUMBAR AND SACRAL REGIONS.

Finally, it should be stated that typical " classical " forms of Brown-Séquard's syndrome are much rarer than atypical, as we seldom have to do with a clean and complete unilateral division, the lesion generally either not amounting to a complete hemisection, or else passing over, and affecting more or less the second side of the cord.

APPENDIX.

The differential diagnosis between intramedullary and extramedullary intravertebral tumours occasionally presents great difficulties. There is no symptom which is pathognomonic of the one while it decisively excludes the other. At the same time the following criteria favour, in general, the diagnosis of an extramedullary as opposed to an intramedullary tumour, and, as is well known, the operative prognosis in extramedullary tumours is steadily improving as the surgery of the nervous system advances:

1. A pseudo-neuralgic prodromal stage preceding symptoms of organic disturbance (root pains). Extramedullary tumours are generally situated on the dorso-lateral spinal periphery.

2. Slow but continuous development of motor and sensory disturbances.

3. Tendency of the motor and sensory disturbances which may be present to remain stationary so far as extension upwards is concerned, while increasing in intensity.

4. Persistence of Brown-Séquard's syndrome.

5. Marked intensity of the spastic symptom complex and persistence thereof after paraplegia is fully established. Early and marked development of Babinski's sign and " flight reflex."

6. Presence of irritative motor symptoms (spastic phenomena, spinal epilepsy).

7. Absence or only slight development of degenerative atrophic paralyses. (The amount of muscular atrophy present is often quite out of proportion to the very slight alterations in electrical excitability.)

8. Absence of trophic changes in the soft tissues.

9. Certain anomalies of the cerebro-spinal fluid. Lumbar puncture shows very often in cases of extramedullary tumour a marked globulin increase with slight or absent lymphocytosis, sometimes also a xanthochromia—*i.e.*, a yellow coloration due to blocking of the circulation distal to the tumour, with or without extravasation of blood-pigment.

10. Sensitiveness of the vertebræ to pressure.

On the other hand, a typical and well-marked dissociated anæsthesia points strongly to an intramedullary situation for the tumour (*vide* p. 29), while a simple prevalence of disturbances of common sensation and the temperature sense, as compared with other sensory disturbances, is not uncommon with extramedullary growths (increased sensitiveness of common sensation and thermal fibres to pressure).

B. "LONGITUDINAL" OR SEGMENTAL REGIONAL DIAGNOSIS.

The problems to be solved by "longitudinal" diagnosis have been already defined on p. 3. We will here, however, preface our presentation of the material facts with some preliminary general statements which should help towards a logical and systematic grouping of those facts, and towards a clear understanding of their physio-pathological bearing.

CHAPTER I.

THE ANATOMICAL AND PHYSIOLOGICAL BASIS OF SEGMENTAL DIAGNOSIS.

As is well known, the whole body of the embryo arises from separate component parts, or somites, ranged one behind the other. The neural tube, later to become the spinal cord, the integument, and the muscular system, all have a similar origin. Each somite, then, contains a neurotome, a dermatotome, and a myotome, which are intimately connected with each other. For each embryonal spinal segment sends, by way of its pair of anterior roots, motor fibres to the corresponding muscle segments, and receives, in like manner, from the corresponding skin segments, the centripetal fibres of its posterior roots.

In the fully developed organism the signs of segmental origin are in most of the organs no longer visible. This applies also to the greater part of the muscular system. From four myotomes—A, B, C, D—lying one behind the other, three muscles—I., II., III.—may, for instance, be so formed that muscle I. shall consist of parts from A, B, C, and D; muscle II. of parts from B and C; and muscle III. of parts from B, C, and D. To take a concrete instance: The biceps femoris is composed of elements from the two last lumbar myotomes and the two first sacral myotomes (L_4, L_5, S_1, S_2); so also the gluteus maximus. The semitendinosus and semimembranosus, on the other hand, are made up of elements from L_4, L_5, and S_1, and the tibialis anticus of elements from L_4 and L_5 only. Consequently, in the developed organism the biceps femoris and the gluteus maximus will receive their motor nerve fibres from the anterior roots of

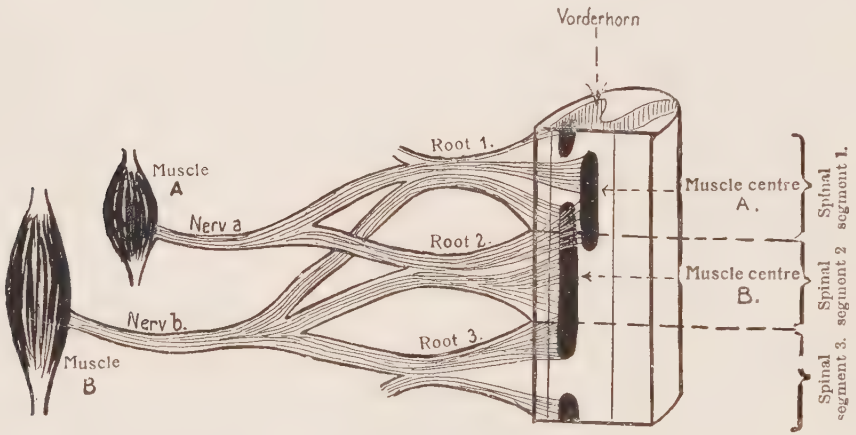


Fig. 14.—Radicular and Peripheral Muscular Innervation.

Muscle A supplied by segments 1 and 2.
Muscle B supplied by segments 1, 2, and 3.

spinal segments L_4—S_2; while the "radicular innervation" of the semimembranosus and semitendinosus is L_4—S_1, and of the tibialis anticus L_4, L_5. As regards the peripheral innervation, however, of the muscles in question, we find that the biceps femoris, semimembranosus, semitendinosus, and tibialis anticus are supplied by the deep peroneal branch of the sciatic; the gluteus maximus, on the contrary, by the inferior gluteal nerve. Both the sciatic and the inferior gluteal, then, draw fibres from the spinal root pairs L_4—S_2, and analogous conditions obtain in the case of all other peripheral motor nerves. Each contains fibres from several anterior roots, and, conversely, each anterior root distributes its fibres among several peripheral nerve trunks. This often exceedingly complicated interchange of fibres takes place in the so-called "plexuses," whose special features are given in every textbook of anatomy.

Here we will merely give a rough diagrammatic representation of the principle of radicular and peripheral motor innervation (Fig. 14).

In the above diagram we see also that the centres for the separate muscles present themselves, in a longitudinal section of the grey matter of the cord, as nuclear columns lying side by side, and one above the other. It is these columns which, in a transverse section, are seen as the cell-groups of the anterior horn, with which we made acquaintance on pp. 12 and 13.

A careful study of the atrophy of spinal nerve cells supervening on amputations, on experimental destruction of nerve fibres, or on decay of single muscles, has given us a knowledge of the position of the more important muscular centres within the ventral horn, which, though by no means complete, is yet fairly sufficient for our clinical requirements.

In general it may be said that the centres for the spinal musculature are to be found in the dorso-mesial group, those for the muscles of the proximal segments of the limbs in the ventro-mesial, while the two lateral groups govern the remaining segments of the extremities. The centres for the coarser movements of flexion and extension are in the neighbourhood of the periphery, while those for the finer movements (*e.g.*, of toes and fingers) lie nearer the central groups. We may here again draw attention to the fact that the latter contain the vaso-motor centres.

Complete paralysis and loss of tone in a muscle may be caused, as is made plain in Fig. 14, not only by a lesion in the cord which destroys its whole spinal nucleus, but also by the destruction of its peripheral nerves. Anterior root lesions, on the other hand, unless very extensive, merely weaken, and do not completely paralyze, the muscle, owing to the fact that, as a rule, the muscle is innervated from several roots. It will be necessary, therefore, to state as clearly as possible the distinguishing marks between radicular and peripheral paralyses.

To return to the plexuses:

We have up to the present considered them with reference only to their motor constituents. As, however, soon after their exit from the cord, the posterior roots unite with the anterior to form mixed nerves, the nerve trunks issuing from a plexus on its distal side will contain, not only contingents from several anterior roots, but also sensory fibres from various spinal ganglia or posterior roots. Further, each separate spinal ganglion, as also each ventral horn centre, sends fibres to several distinct nerve trunks. There is this marked distinction, however: while the muscle acts as a functional unit, so that we do not find portions thereof paralyzed in consequence of lesions affecting some only of its allotted anterior roots, we meet with quite different conditions when we come to consider the innervation of the integument.

Notwithstanding the often intricate routes which they have followed along the peripheral nerve trunks, the sensory fibres are always distributed

to the extreme periphery in such manner that the "complex" entering a given posterior root supplies with afferent fibres a definite integumental area. Such area is termed a "radicular zone" or "root field." In it are reunited the

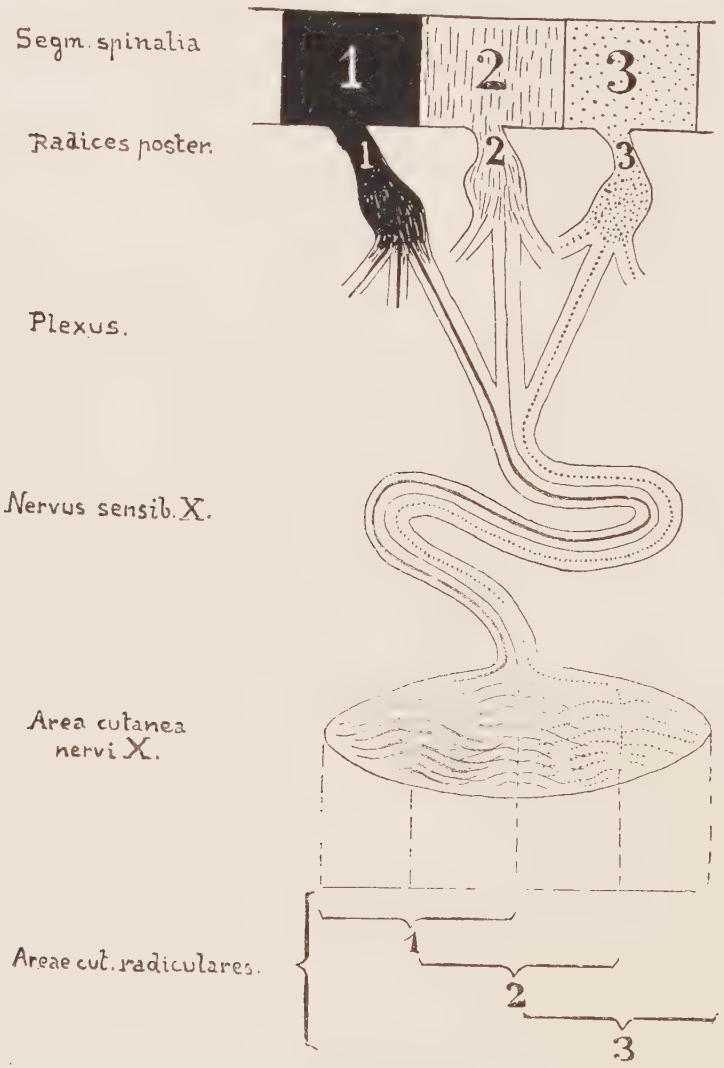


FIG. 15.—RADICULAR AND PERIPHERAL CUTANEOUS INNERVATION.
Note the overlapping of the contiguous root fields.

sensory fibres, which were originally united in a posterior root, though they may have found their way to the periphery along several nerve trunks. It might naturally be inferred from the foregoing that the destruction of a single posterior root would cause an integumental anæsthesia of sharply defined

boundaries. This is not, however, the case, inasmuch as the separate radicular zones overlap each other somewhat like the tiles of a roof, and thus each portion of the integument is innervated from two posterior roots. Only when two adjacent posterior roots or spinal ganglia are destroyed does a "radicular" anæsthesia supervene. If, however, a peripheral nerve is severed, we have an integumental anæsthesia of quite different distribution, of so-called "peripheral" type. The anæsthesia affects the whole area of distribution of the injured nerve trunk, and is made up, after the manner of a mosaic, of those portions of separate "root" zones which go to the composition of the nerve trunk in question. It is obvious, therefore, that the conditions underlying radicular are far simpler than those underlying peripheral innervation of the integument (*vide* Fig. 15).

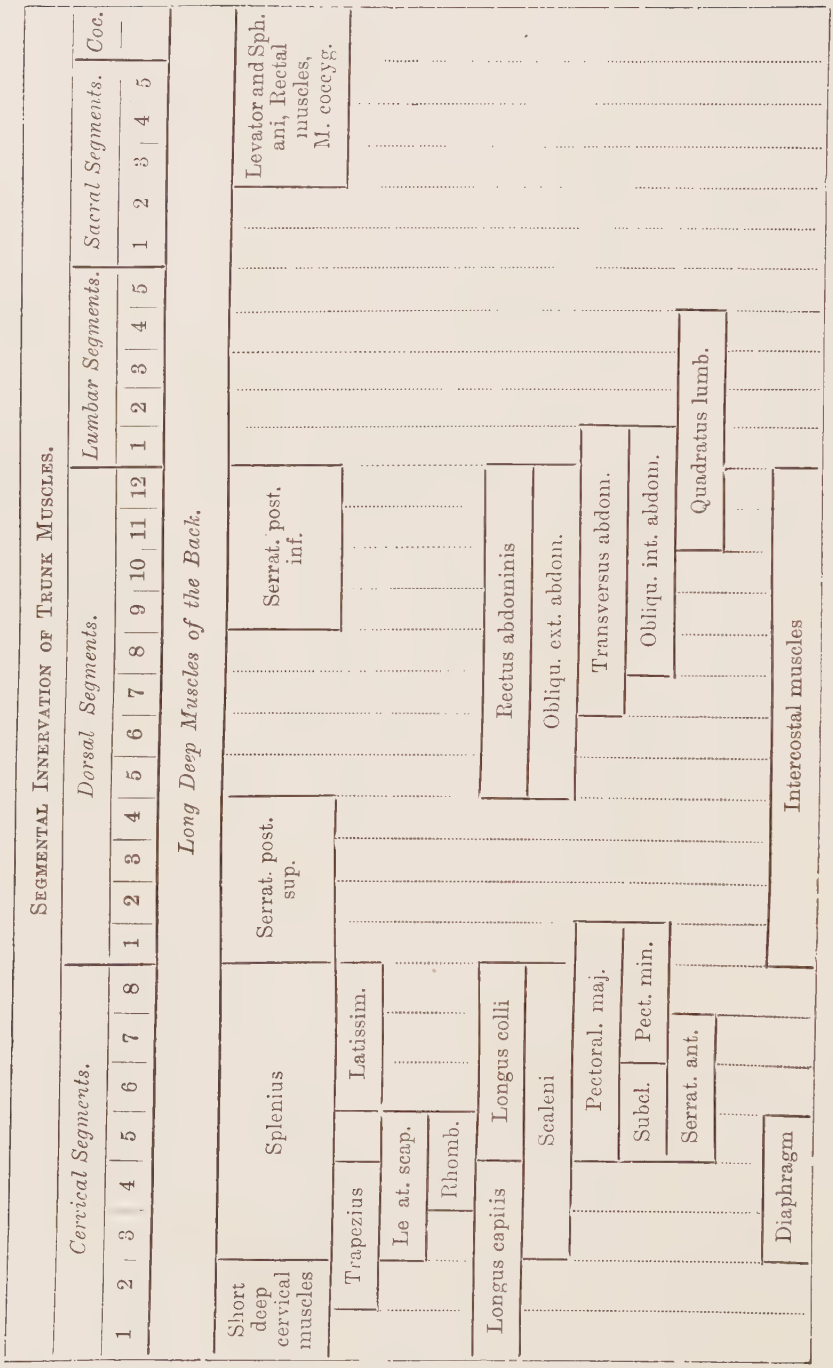
CHAPTER II.

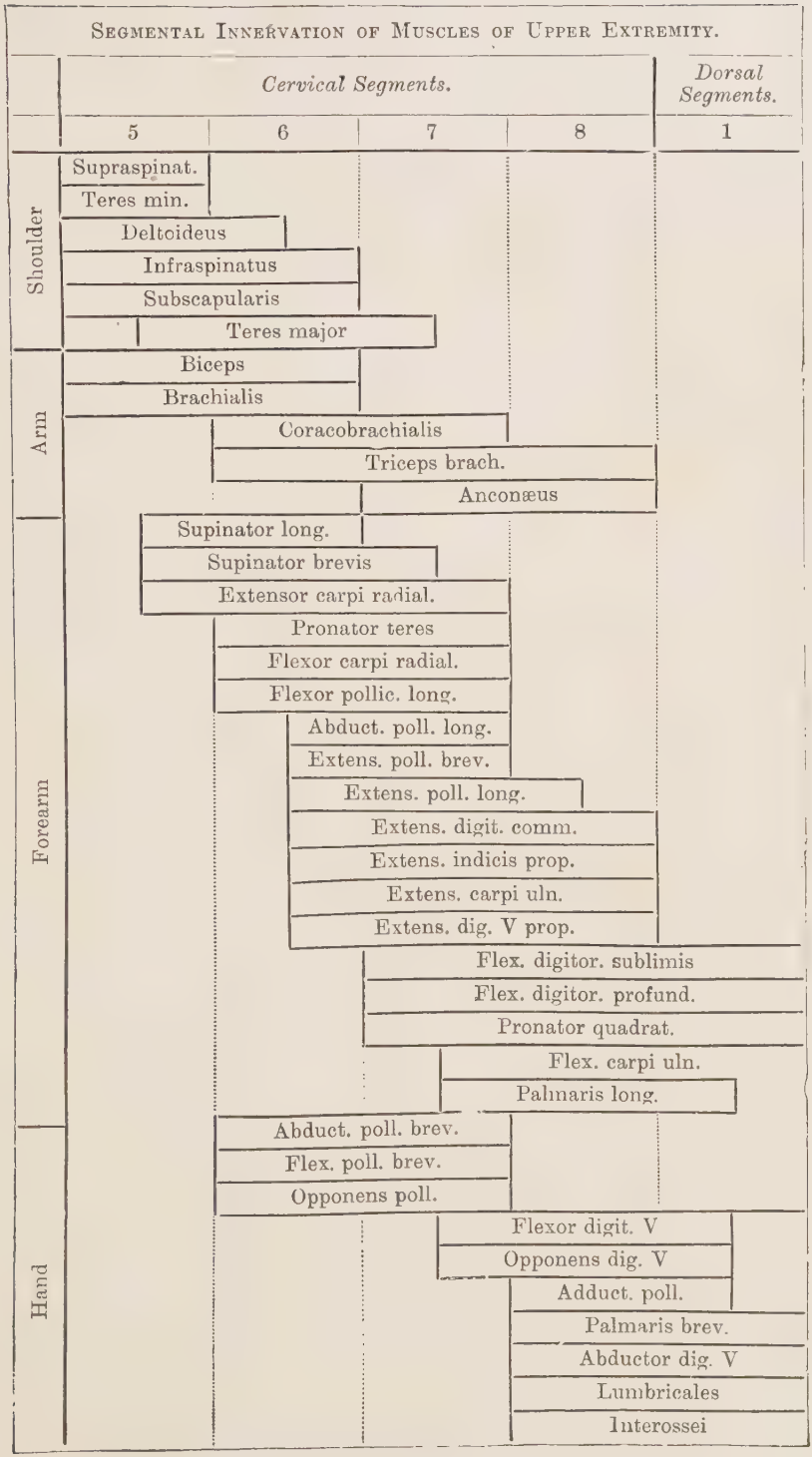
I. SEGMENTAL DIAGNOSIS IN MOTOR PARALYSES. DIFFERENTIAL DIAGNOSIS BETWEEN RADICULAR AND PERIPHERAL PARALYSES.

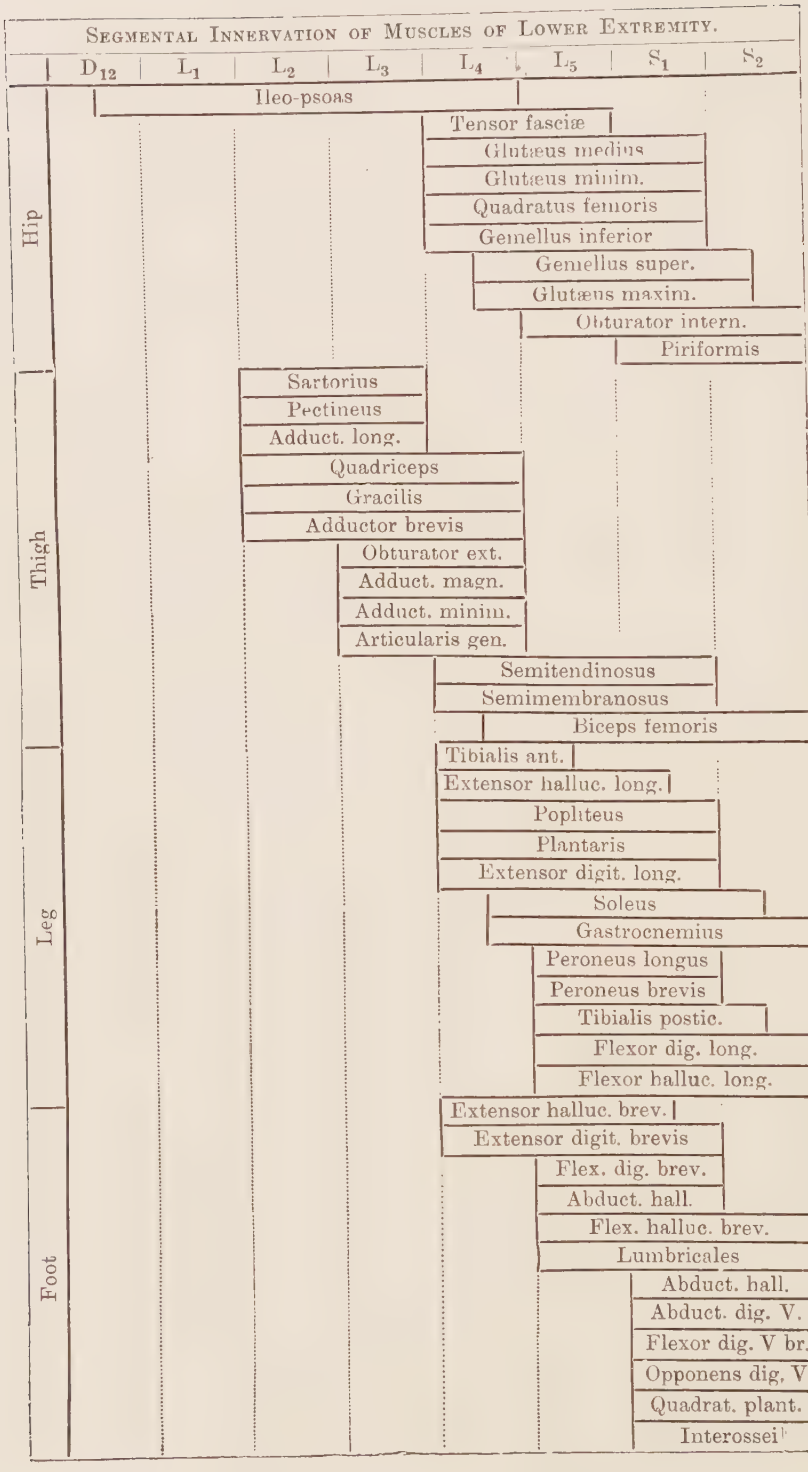
After the preliminary remarks in the foregoing chapter, the tables on pages 44, 45, 46, in which the radicular innervation of the muscles is given, require no comment.

It is easy to see at a glance from the foregoing tables what muscles are likely to be paralyzed by a lesion affecting the ventral horns or anterior roots in any given segments of the cord. To distinguish these "radicular" paralyses from others of peripheral origin (from such, that is to say, as are caused, not by the destruction of motor roots, but by that of peripheral nerve trunks and branches), it is necessary to bear in mind also the conditions of peripheral innervation, as given in the following tables. In the first column we have given the separate peripheral nerves, in the second the muscles supplied by them, while in the third are given the movements carried out by the respective muscles. Ability or inability to carry out its appropriate movements points, respectively, to the functional soundness or to the paralysis of the muscle under consideration. The power of voluntary as well as of electrically excited contraction must be tested. For the latter purpose a knowledge of the electrical stimulation points given in all works on electro-diagnosis is essential.

The following point is now clear: If we have a simultaneous paralysis affecting a group of muscles which are supplied by the same peripheral nerves, we may infer a peripheral lesion; if, on the other hand, the group of muscles affected is one possessing the same radicular innervation, a spinal lesion is indicated.


Segmental Innervation of Trunk Muscles.
Cervical Segments.
1 2 3 4 5 6 7 8
Dorsal Segments.
1 2 3 4 5 6 7 8 9 10 11 12
Lumbar Segments.
1 2 3 4 5
Sacral Segments.
1 2 3 4 5
Coc.
—
Long Deep Muscles of the Back.
Short deep cervical muscles
Splenius
Serrat. post. sup.
Serrat. post. inf.
Levator and Sph. ani, Rectal muscles, M. coccyg.
Trapezius
Latissim.
Le at. scap.
Rhomb.
Longus capitis
Longus colli
Scaleni
Pectoral. maj.
Subcl.
Pect. min.
Serrat. ant.
Diaphragm
Rectus abdominis
Obliqu. ext. abdom.
Transversus abdom.
Obliqu. int. abdom.
Quadratus lumb.
Intercostal muscles


Segmental Innervation of Muscles of Upper Extremity.
Cervical Segments.
Dorsal Segments.
5
6
7
8
1
Shoulder
Supraspinat.
Teres min.
Deltoideus
Infraspinatus
Subscapularis
Teres major
Arm
Biceps
Brachialis
Coracobrachialis
Triceps brach.
Anconæus
Forearm
Supinator long.
Supinator brevis
Extensor carpi radial.
Pronator teres
Flexor carpi radial.
Flexor pollic. long.
Abduct. poll. long.
Extens. poll. brev.
Extens. poll. long.
Extens. digit. comm.
Extens. indicis prop.
Extens. carpi uln.
Extens. dig. V prop.
Flex. digitor. sublimis
Flex. digitor. profund.
Pronator quadrat.
Flex. carpi uln.
Palmaris long.
Hand
Abduct. poll. brev.
Flex. poll. brev.
Opponens poll.
Flexor digit. V
Opponens dig. V
Adduct. poll.
Palmaris brev.
Abductor dig. V
Lumbricales
Interossei


Segmental Innervation of Muscles of Lower Extremity.
D12
L1
L2
L3
L4
L5
S1
S2
Hip
Thigh
Leg
Foot
Ileo-psoas
Tensor fasciæ
Glutæus medius
Glutæus minim.
Quadratus femoris
Gemellus inferior
Gemellus super.
Glutæus maxim.
Obturator intern.
Piriformis
Sartorius
Pectineus
Adduct. long.
Quadriceps
Gracilis
Adductor brevis
Obturator ext.
Adduct. magn.
Adduct. minim.
Articularis gen.
Semitendinosus
Semimembranosus
Biceps femoris
Tibialis ant.
Extensor halluc. long.
Popliteus
Plantaris
Extensor digit. long.
Soleus
Gastrocnemius
Peroneus longus
Peroneus brevis
Tibialis postic.
Flexor dig. long.
Flexor halluc. long.
Extensor halluc. brev.
Extensor digit. brevis
Flex. dig. brev.
Abduct. hall.
Flex. halluc. brev.
Lumbricales
Abduct. hall.
Abduct. dig. V.
Flexor dig. V br.
Opponens dig. V
Quadrat. plant.
Interossei

A. Plexus Cervicalis (C_1—C_4)		
Nervi cervicales	Musculi profundi colli	Flexion, extension, and rotation of the neck
	Mm. scaleni	Elevation of ribs (inspiration).
N. phrenicus	Diaphragma	Inspiration
B. Plexus Brachialis (C_5—D_2)		
N. thoracic. ant.	M. pect. maj. et min.	Adduction and forward depression of the arm
N. thoracic. long.	M. serrat. ant. maj.	Fixation of the scapula during elevation of the arm
N. dorsalis scap.	M. levator scapul.	Elevation of the scapula
	Mm. rhomboidei.	Elevation and drawing inwards of the scapula
N. suprascap.	M. supraspinatus	Elevation and external rotation of the arm
	M. infraspinatus	External rotation of the arm
N. subscapul.	M. latissimus dors. }	{ Internal rotation and dorsal
	M. teres major }	{ adduction of the arm
	M. subscapularis	Internal rotation of the arm
N. axillaris s. circumflexus	M. deltoideus	Elevation of the arm to the horizontal
	M. teres minor	External rotation of the arm
N. musculocut.	M. biceps brach.	Flexion and supination of the forearm
	M. coraco-brachialis	Flexion and adduction of the forearm
	M. brachialis int.	Flexion of the forearm
N. medianus	M. pronator teres	Pronation
	M. flexor carpi rad.	Flexion and radial flexion of the hand
	M. palm. long.	Flexion of the hand
	M. flex. digit. sublim.	Flexion of the middle phalanges of the fingers
	M. flex. poll. long.	Flexion of the terminal phalanx of the thumb
	M. flex. digit. prof. (radial portion)	Flexion of the terminal phalanges of the index and middle fingers
	M. abduct. poll. brev.	Abduction of the first metacarpal
	M. flex. poll. brev.	Flexion of the first phalanx of the thumb
	M. opponens poll.	Opposition of the first metacarpal
N. ulnaris	M. flexor carpi uln.	Flexion and ulnar flexion of the hand
	M. flex. digit. prof. (ulnar portion)	Flexion of the terminal phalanges of the ring and little fingers
	M. adductor poll.	Adduction of the first metacarpal
	Mm. hypothenaris	Abduction, opposition, and flexion of the little finger
	Mm. lubricales	Flexion of the first phalanges, extension of the others
	Mm. interossei	The same; in addition, spreading and closing together of the fingers
Nervus radialis	M. triceps brach.	Extension of the forearm
	M. supin. longus	Flexion* of the forearm

* The name "supinator longus" is incorrect, inasmuch as electrical excitation experiments show that the muscle has no supinating, but, on the contrary, a slight pronating action. For this reason a preferable name is that of "brachio-radialis." This is in common use among anatomists, but has not yet been adopted by clinicians.

Nervous radialis	M. extensor carpi radi.	Extension and radial flexion of the hand
	M. extensor digit. comm.	Extension of the first phalanges of the fingers
	M. extensor digit. V prop.	Extension of the first phalanx of the little finger
	M. extensor carpi uln.	Extension and ulnar flexion of the hand
	M. supinator brevis	Supination of the forearm
	M. abduct. poll. longus	Abduction of the first metacarpal
	M. extensor poll. brevis	Extension of the first phalanx of the thumb
	M. extensor poll. longus	Abduction of the first metacarpal and extension of the terminal phalanx of the thumb
	M. extensor indic. prop.	Extension of the first phalanx of the index finger
C. Nervi Thoracales	Mm. thoracis et abdominalis	Elevation of the ribs, expiration, compression of abdominal viscera, etc.
D. Plexus Lumbalis (D_{12}—L_4)		
Nerv. cruralis	M. ileo-psoas	Flexion of the hip
	M. sartorius	Internal rotation of the leg
	M. quadriceps	Extension of the leg
Nerv. obturatorius	M. pectineus M. adductor longus M. adductor brevis M. adductor magnus M. gracilis	Adduction of the thigh
	M. obturator extern.	Adduction and external rotation of the thigh
E. Plexus Sacralis (L_5—S_5)		
N. glutæus sup.	M. glutæus med. M. glutæus min.	Abduction and internal rotation of the thigh
	M. tens. fasciæ latæ	Flexion of the thigh
	M. pyriformis	External rotation of the thigh
N. glutæus inf.	M. glutæus max.	Extension of the thigh
N. ischiadicus	M. obturator int. Mm. gemelli M. quadratus fem.	External rotation of the thigh
	M. biceps femoris M. semitendinosus M. semimembranosus	Flexion of the leg
(*a*) N. peroneus: (*a*) Prof.	M. tibialis ant.	Dorsal flexion and supination of the foot
	M. extens. digit. long.	Extension of the toes
	M. extens. hall. long.	Extension of the great toe
	M. extens. digit. brev.	Extension of the toes
	M. extens. hall. brev.	Extension of the great toe
(*b*) Superf.	Mm. peronei	Dorsal flexion and pronation of the foot
(*β*) N. tibialis	M. gastrocnemius M. soleus	Plantar flexion of the foot
	M. tibialis post.	Adduction of the foot
	M. flex. digit. long.	Flexion of the terminal phalanges, II—V
	M. flex. halluc. long.	Flexion of the terminal phalanx of the great toe
	M. flex. digit. brev.	Flexion of the middle phalanges, II—V
	M. flex. halluc. brev.	Flexion of the first phalanx of the great toe

(β) N. tibialis	Musculi plantares pedis reliqui	Spreading and closing together of the toes and flexion of the first phalanges
N. pudendus	Mm. perinei et sphincteres	Closing of the pelvic organs, co-operation in the sexual act

In Figs. 16 to 23 we have represented the more important muscles of the extremities in diagrammatic transverse sections of the arm and forearm, thigh

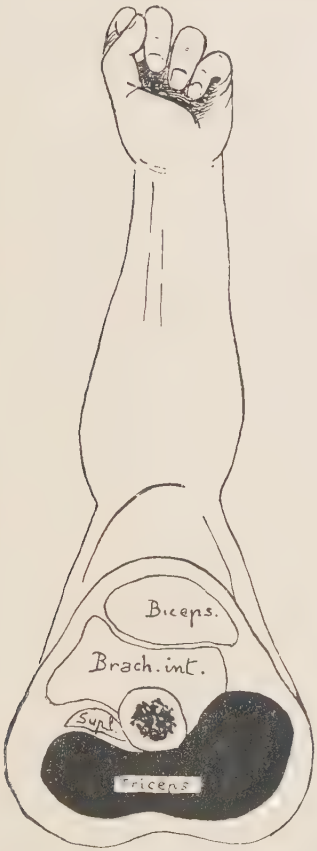


FIG. 16.—RADICULAR INNERVATION OF THE MUSCLES OF THE ARM.
White, C_5—C_6; black, C_6—C_8.

FIG. 17.—PERIPHERAL INNERVATION OF THE MUSCLES OF THE ARM.
White=musculo-cutaneous; black=radial.

and leg, and by distinctive shading, etc., have grouped the muscles in accordance with their radicular and peripheral innervation respectively.

The following special examples may be cited: In paralysis of the radial nerve in the forearm the extensors of the wrist and of the fingers are affected in company with the supinator longus; while, on the contrary, in the so-called "forearm type" of spinal paralysis, flexors and extensors of the wrist and fingers are affected in common, and the supinator longus generally escapes,

because its radicular innervation reaches upwards to the fifth cervical segment. Again, paralysis involving the deltoid and teres minor alone is a somewhat frequently observed affection, pointing to disease or injury of a peripheral nerve, its relative frequency being due to the exposed position of the affected nerve, the circumflex, in its course round the humerus. Cases are also met with in which the deltoid only is affected, the teres minor escaping owing to the fact that it receives fibres also from the suprascapular nerve. This circumscribed paralysis is never the result of a spinal lesion. In the corresponding

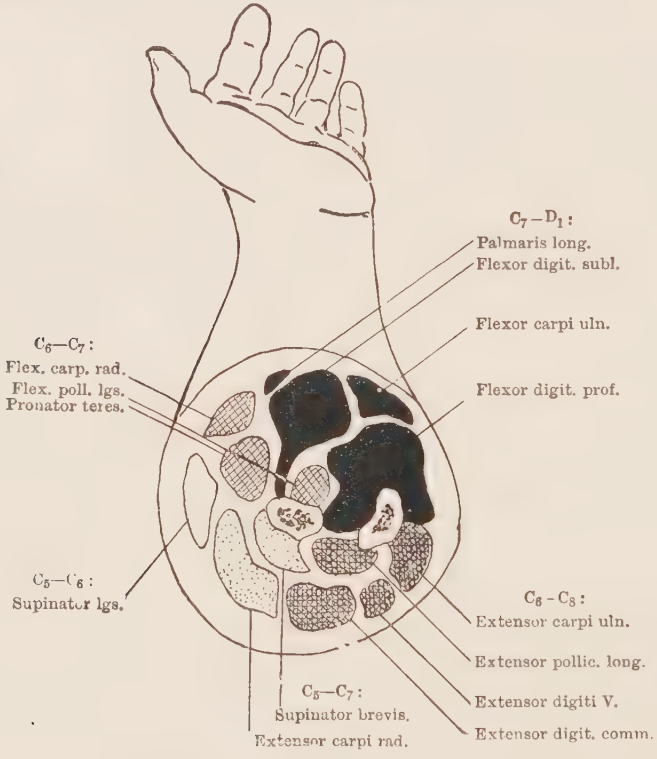


FIG. 18.—RADICULAR INNERVATION OF FOREARM MUSCLES.

form of paralysis of spinal origin, the "upper arm type," the scapular muscles, the biceps, and the supinator longus, are also affected.

This "upper arm type" of paralysis may be produced, not only by a radicular lesion affecting the fifth and sixth cervical roots, but also by a lesion of the primary plexus trunk which is formed by a union of these two roots, and which is, owing to its superficial situation, extremely liable to injury. This form is spoken of as "Duchenne-Erb plexus paralysis."

By analogy with the above-named types, one speaks, in discussing radicular paralyses, of "hand," "thigh," "leg," and "foot" types, according to the limb segment chiefly affected.

Fig. 24 represents a radicular paralysis of fingers following a traumatic lesion of the seventh and eighth cervical roots. The paralysis involves parts of the musculature innervated by the radial as well as that by the ulnar nerve. Fig. 25 serves for comparison. It represents a peripheral ulnar paralysis, also of traumatic origin.

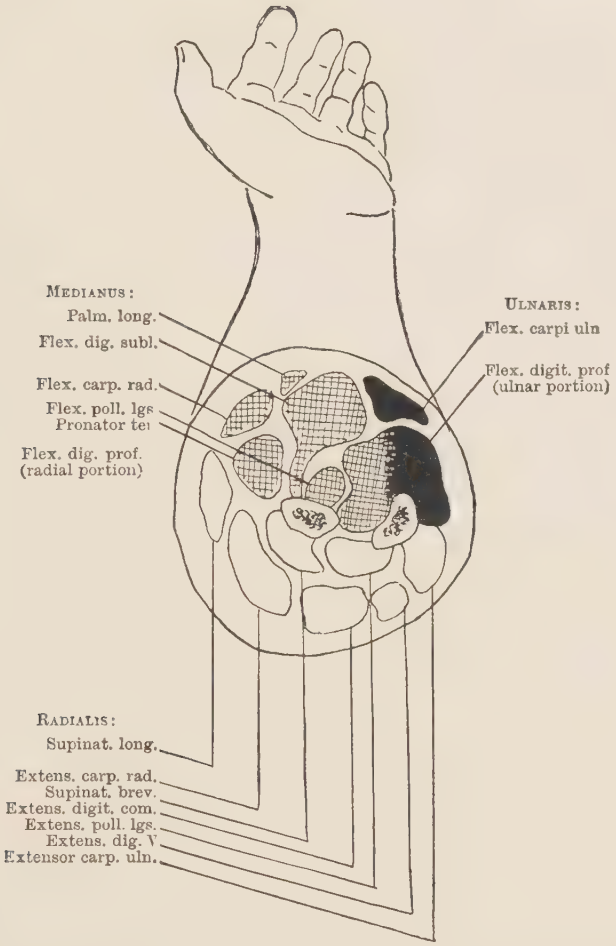


Fig. 19.—Peripheral Innervation of Forearm Muscles.

Where we have to deal with a lesion not merely affecting at a given point in the cord the spinal muscular centres—*i.e.*, the ventral horns—but also the cortico-spinal neurons, we shall find paralyzed, not only those muscles which are innervated from the injured roots, but also all those innervated from anterior roots distal to the site of the lesion. From the tables given on pp 44-46 one may, for any given transverse lesion, read off the complex of

muscles that will be paralyzed by taking all those to the right of, or included in, the column of the affected segment. For rapid reference the following table is added, in which the different spinal levels and the motor mechanisms respectively subject thereto are given in summary form:

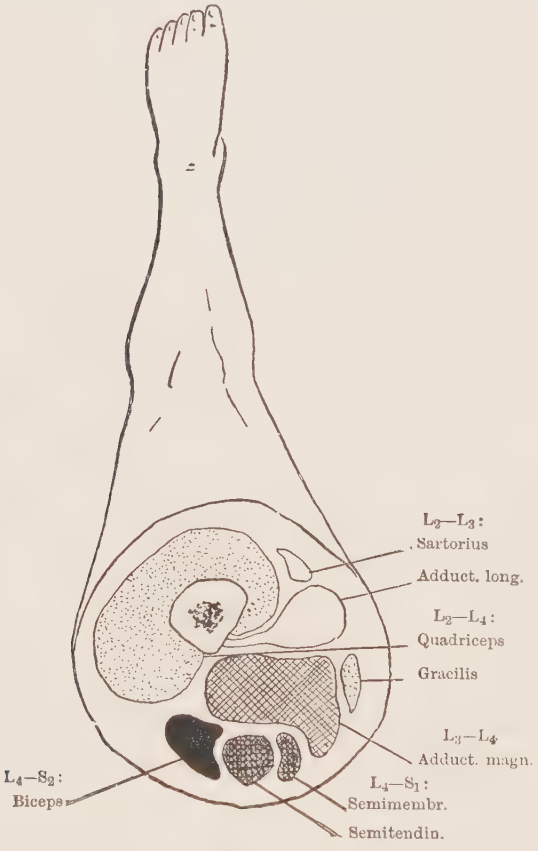


FIG. 20.—RADICULAR INNERVATION OF THIGH MUSCLES.

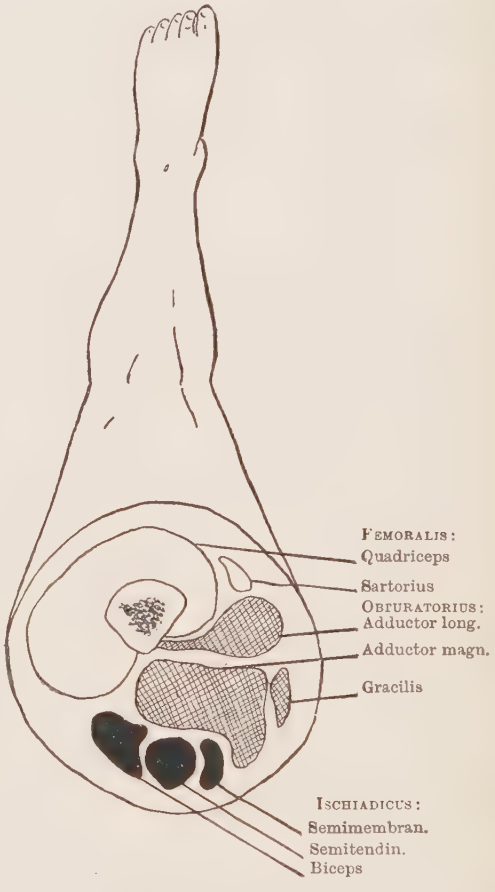


FIG. 21.—PERIPHERAL INNERVATION OF THIGH MUSCLES.

Cervical Cord.			*Dorsal Cord.*	*Lumbar Cord.*		*Sacral Cord.*
Upper.	*Middle.*	*Lower.*		*Upper.*	*Lower.*	
Movements of head and neck. Elevation of shoulders	Diaphragmatic breathing. Movements of arm. Movements of forearm	Movements of hand and fingers	Intercostal muscles. Abdominal muscles	Flexion at hip-joint. Adduction of thigh	Movements of thigh (except adduction). Movements of leg	Movements of feet and toes, spincters, rectal muscles

In the classical cases of fracture or dislocation of the vertebral column, as also in Pott's disease, importance from a clinical and diagnostic point of view attaches, not only to the muscle complex involved in the paralysis, but also to the condition of irritability affecting the parts innervated from the region of the cord immediately above the lesion.

We may have, for instance, functional hyperactivity of particular spinal centres, contracture of the muscles innervated from them, and extremely

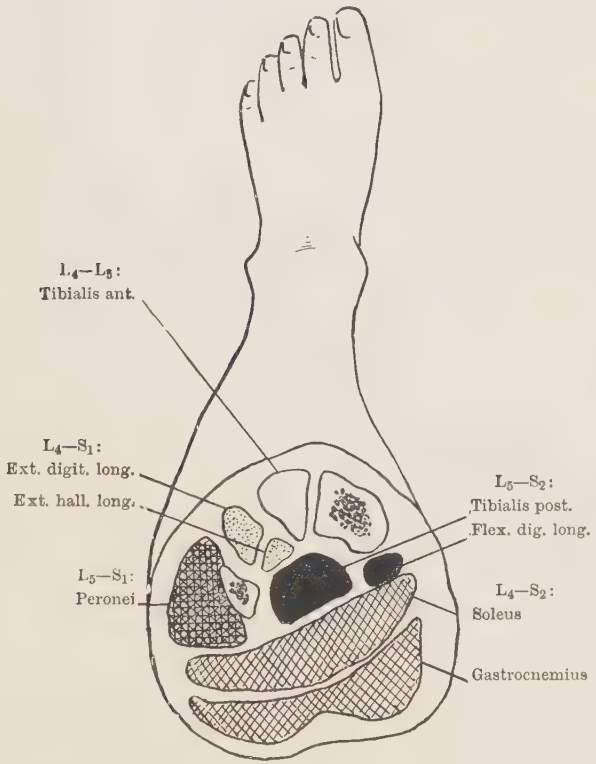


FIG. 22.—RADICULAR INNERVATION OF LEG MUSCLES.

characteristic forced positions of the extremities. Where antagonist muscles suffer simultaneously from irritative contracture, the originally more powerful muscles prevail. A few examples will illustrate these points.

A transverse lesion at the first dorsal segment may cause clawlike contracture of the hand, the mobility of the upper extremity remaining otherwise perfect. This is due to "irritation" of the flexors of the fingers, which are innervated chiefly from the last cervical segment. In a lesion of that segment we have as a rule a forced semiflexion of the forearm, due to irritation of the

biceps and brachialis centres, which lie immediately above the seat of the lesion (*vide* Fig. 26). Similarly, the forced abduction and external rotation of the arm, which is often noticed in association with a lesion of the sixth cervical segment, is explained as being due to a condition of irritation affecting the immediately proximal centres for the supraspinatus, teres minor, deltoid, infraspinatus, etc. A specially typical case of the kind is the spastic flexion of the

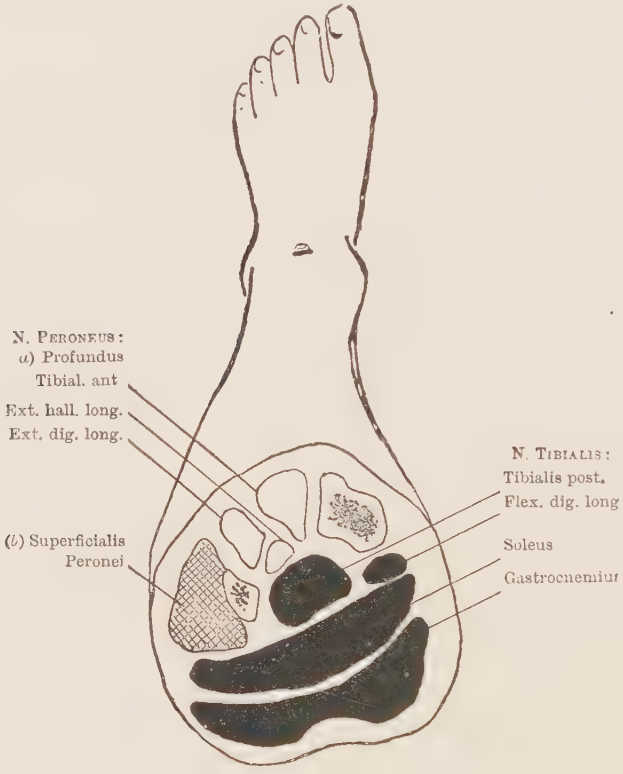


Fig. 23.—Peripheral Innervation of Leg Muscles.

hip (irritation of the ilio-psoas centres) in lesions of the lower lumbar cord (*vide* Fig. 27).

In incomplete transverse lesions of the cervical cord, the paraplegia of the arms is sometimes more severe than that of the lower limbs. This is due to the fact that, in the lateral columns, the pyramid fibres for the upper extremities are more external in position, and consequently more exposed to injury, than those for the lower.

II. SEGMENTAL DIAGNOSIS IN SENSORY DISTURBANCES, AND DIFFERENTIAL DIAGNOSIS BETWEEN RADICULAR AND PERIPHERAL DISTURBANCES OF SENSATION.

Still more significant than in the case of motor disturbances is the distinction between sensory disturbances of peripheral and those of spinal or radicular causation. Figs. 28 and 29, in which are given, on one side the integumental innervation areas of the separate nerve trunks, on the other the sensory "root fields," or radicular areas, make this distinction and its importance plain at a glance. In connection with the representation of the root

FIG. 24.—TRAUMATIC RADICULAR PARALYSIS OF RING AND LITTLE FINGERS.

fields in the figures, it is to be borne in mind that the radicular areas extend on either side of the line bearing the corresponding segment numbers. It is plain from this that an area of anæsthesia is only to be demonstrated clinically when at least two contiguous posterior roots are involved. Most of the diagrams of segment innervation of the integument given in works on neurology leave the above very important fact out of consideration, and represent the separate radicular areas as ribbon-like and sharply defined. I have adhered to the Edinger schema, which gives much the best representation of the actual conditions.

In undertaking operative measures, the site of the spinal lesion must

always be taken to be a segment higher than would be concluded from the upper limit of the area of total anæsthesia, if the overlapping of the root fields were left out of account.

The special type of arrangement of the radicular zones in the extremities, in which, in contrast to the circular arrangement in the trunk, a longitudinal distribution prevails, is explained by reference to ontogenetic conditions. In the embryo the extremities, as they sprout from the trunk, draw with them the dermatotomes lying within their range, and as the limbs develop more or less perpendicularly to the axis of the trunk, the circular arrangement must, in them, give place to the longitudinal.

It may be mentioned here that in lesions of the spinal ganglia an eruption of herpes zoster frequently breaks out on the radicular area of the affected posterior roots. No satisfactory explanation of this phenomenon has as yet been given.

III. SEGMENT DIAGNOSIS IN REFLEX DISTURBANCES.

Reflex disturbances, as well as paralyses and disturbances of sensation, play an important part in the regional diagnosis (longitudinal) of spinal affections. Reflexes are abolished at the level at which the "reflex arc" is broken. By co-ordination of clinical and anatomico-pathological observations, we have

Fig. 25.—Traumatic Peripheral Ulnar Paralysis.

arrived at a fairly exact knowledge concerning the levels of the separate reflex arcs. The following table gives in compact form the present state of our knowledge on the subject. It is only in quite young individuals that one can expect to be able to demonstrate all the reflexes. After puberty the great majority of the reflexes given in the table cannot be elicited even in perfectly healthy individuals.

	Tendon and Bone Reflexes.	*Skin Reflexes.*	*Method of Starting.*	*Effect.*	*Localization.*
1	—	Scapular reflex	Stimulation of the skin over the scapula	Contraction of shoulder-blade muscles	C_5—D_1
2	Biceps reflex	—	A blow on the biceps tendon	Flexion of forearm	C_5—C_6
3	Triceps reflex	—	A blow on the triceps tendon	Extension of forearm	C_6—C_7
4	Scapulo-humeral reflex	—	A blow on the inner side of the lower angle of the scapula	Adduction of arm	C_6—C_7
5	Radius reflex	—	A blow on the styloid process of the radius	Supination of forearm	C_7—C_8
6	—	Palmar reflex	Irritation of the palm	Flexion of fingers	C_8—D_1
7	—	Epigastric reflex	Stroking from nipple downwards	Drawing in of epigastrium	D_7—D_9
8	—	Upper abdominal reflex	Stroking skin of upper part of abdomen	Drawing in of abdominal wall	D_8—D_9
9	—	Mid abdominal	Stroking skin of middle and lower parts of abdomen	Drawing in of abdominal wall	D_{10}—D_{12}
10	—	Lower reflex			
11	—	Cremaster reflex	Stroking the adductor region of the thigh	Elevation of testis	L_1—L_2
12	Patellar reflex	—	A blow on the quadriceps tendon	Extension of leg	L_2—L_4
13	—	Gluteal reflex	Stroking the nates	Contraction of glutei	L_4—L_5
14	Achilles reflex	—	A blow on the Achilles tendon	Flexion of foot	S_1—S_2
15	—	Plantar reflex	Stroking of the sole	Flexion of toes	S_1—S_2
16	—	Anal reflex	Pricking the perineum	Contraction of sphincter ani externus	S_5

The patellar reflex is constant under normal conditions, and the Achilles reflex almost equally so. Much less constant are the skin reflexes, of which, however, the cremaster, abdominal, and plantar reflexes can be elicited in the great majority of healthy individuals. The utility of the skin reflexes from the point of view of localization is, however, much diminished by the fact that they may be abolished as the result of lesions situated far above the level generally accepted as that of the corresponding reflex arcs—as the result, for instance, even of cerebral lesions. It is therefore generally admitted that the mechanism of skin reflexes is far more complicated than that of tendon reflexes. The centripetal fibres of the abdominal reflex, for instance, enter the lower dorsal cord by way of certain posterior roots, and its centrifugal fibres leave the cord by way of corresponding anterior roots. In the majority of individuals, however, the irritative impulse is perhaps not conveyed by

the direct reflex arc, as depicted in Fig. 7, but by the intervention of connecting fibres (*cf.* pp. 18 and 19) which pass some distance, first in a frontal, and then, again, in a caudal, direction. This hypothesis would serve to explain how it is

FIG. 26.—FORCED POSITION OF THE ARMS IN LESION OF THE SEVENTH CERVICAL SEGMENT.

that a skin reflex may be abolished, not only by a lesion at its corresponding segment level, but also in certain circumstances by one at a much higher level.

FIG. 27.—FLEXION OF THE HIPS IN LESION OF THE LOWER LUMBAR CORD.

We cannot at present put forward further conjectures concerning this very complicated mechanism; it is a subject which calls urgently for further elucidation.

Further, skin reflexes are occasionally elicited by irritation of quite atypical areas—the cremaster reflex, for instance, by irritation of the sole of the foot.

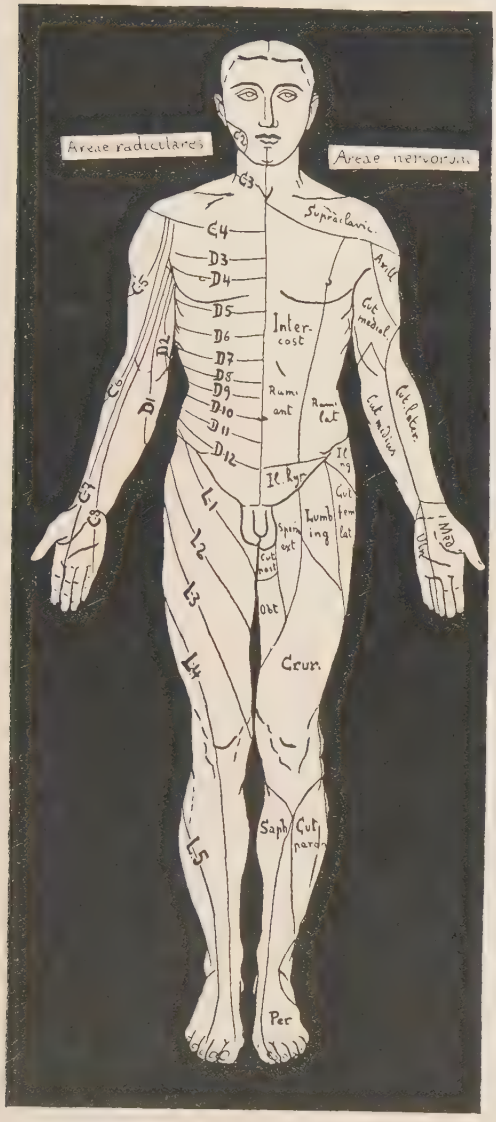


FIG. 28.

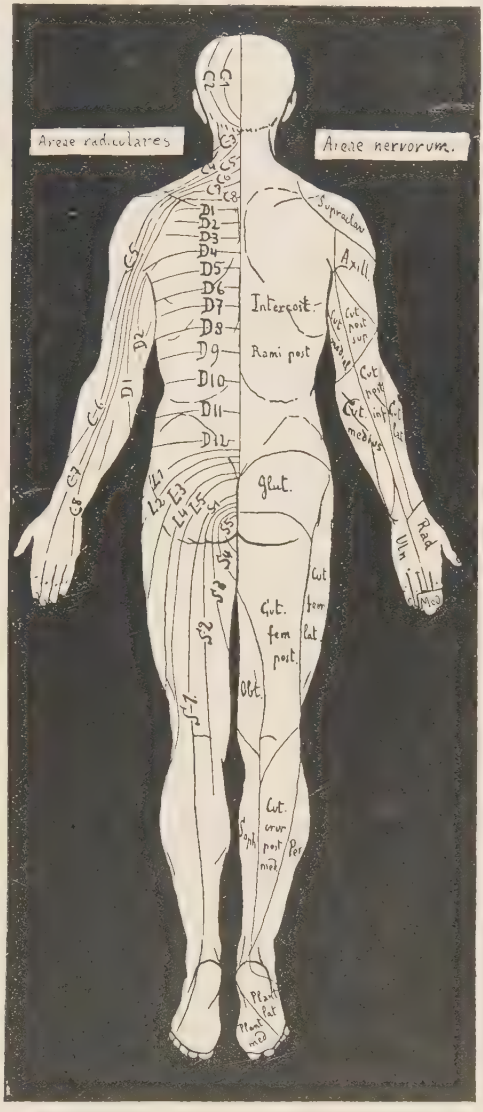


FIG. 29.

The vaso-motor reflex mentioned on p. 22 (dermographia spinalis or dolorosa, "reflex erythema") can occasionally be employed in segment diagnosis, as, for instance, when it is absent from the radicular zone corresponding to the site of a spinal lesion, but is easily demonstrated above and below that area. The same applies to the phenomenon known as "goose skin."

CHAPTER III.

It remains for us, before closing our consideration of the principles of regional diagnosis in spinal affections, to discuss certain special symptom-complexes which give to lesions of the cervical and upper dorsal cord on the one hand, and to those of the conus terminalis, together with the cauda equina, on the other, quite a special character, and one, consequently, of great importance from the point of view of regional diagnosis.

I. DIAGNOSTIC CHARACTERS OF LESIONS AFFECTING THE UPPER PART OF THE SPINAL CORD.

(*a*) Oculo-Pupillary Symptoms.*

The development of these phenomena in lesions of the cervical and upper dorsal regions is easily intelligible if we bear in mind the anatomical and physiological conditions of the parts (*vide* Fig. 31).

The upper part of the sympathetic chain is represented, as is well known, by the three sympathetic cervical ganglia (ganglion cervicale superius, medium, and inferius). In the last named are motor cells whose cranially directed axis cylinders innervate the following muscles:

1. The dilator pupillæ.

2. The unstriped (involuntary) portion of the levator palpebræ superioris (musculus tarsalis superior).

3. The unstriped (involuntary) musculus orbitalis. This (the remains of a powerfully developed muscular layer which is found in those mammals whose orbits have a wide communication with the temporal fossa) passes across the lower orbital fissure, and prevents the contents of the orbit from sinking backwards.

These three sections of motor fibres are under the innervatory influence of a nuclear group of cells situated in the lateral horn of the last cervical and upper dorsal segments, the centrum cilio-spinale. The connection is made through the eighth cervical and first and second dorsal roots and their rami communicantes.

The centrum cilio-spinale itself is under the influence (probably through the lateral column) of a bulbar centre, concerning the exact anatomy of which we have, it is true, no certain knowledge. That this centre, however (so far, at any rate, as its dilating neurons are concerned), is governed from the cortex cerebri is deduced, not only from experimental observations, but also from

* A combination of these oculo-pupillary symptoms with the vaso-motor secretory symptoms dealt with on pp. 63, 64, is known as "Horner's symptom complex" (*cf.* Fig. 31).

observation of the dilatation of the pupil which accompanies feelings of terror, pain, the sexual orgasm, etc. There is even an ideo-motor mydriasis, which may be brought about by a very vivid mental conception of darkness. Thus, between the sympathetic oculo-pupillary mechanism and the vaso-motor mechanism described on pp. 20 and 21 there is a close agreement in principle. In the one case, as in the other, we have centres which may be ranged in four orders, with cerebro-bulbar, bulbo-spinal, spino-sympathetic, and sympathetico-muscular connecting routes.

Interruption of the sympathetic oculo-pupillary innervation announces itself, as will be readily understood from the foregoing—

1. By a paralytic myosis (spinal myosis), in which the pupil, contracted, owing to paralysis of the dilating fibres, no longer dilates, even when the eye is shaded from the light. The inequality of the pupils (anisocoria) which accompanies unilateral lesions of this mechanism is, therefore, much more easily demonstrated in partial darkness than in a bright light, as, in the latter case, the antagonizing sphincter pupillæ innervated by the oculo-motor nerve comes into action on both sides.

2. By a narrowing of the space between the eyelids, so-called "sympathetic ptosis," due to paralysis of the unstriped elevator muscle, the superior tarsal.

Fig. 30.—Sympathetic Oculo-Pupillary Symptom Complex in Klumpke's Paralysis.

3. In some cases by a sinking of the eyeball into the orbit (enophthalmos). This symptom is due to paralysis of the orbital muscle. It is clear that the foregoing symptoms may be brought about by several different conditions—*e.g.:*

(α) By lesions in the region of the cervical sympathetic.

(β) By injury or disease in the region of the centrum cilio-spinale (lowest cervical and upper dorsal cord).

(γ) By lesions of the lowest cervical and the two upper dorsal anterior roots and their rami communicantes. The sympathetic oculo-pupillary

symptom complex may therefore be an accompaniment of the so-called "lower brachial plexus paralysis," or *Klumpke's* paralysis. This latter is due to a lesion (the result of neoplasm, syphilitic meningitis, injury, etc.) of the eighth cervical and first dorsal roots, and its motor manifestations involve the small muscles of the hand and the flexors of the forearm. The oculo-pupillary

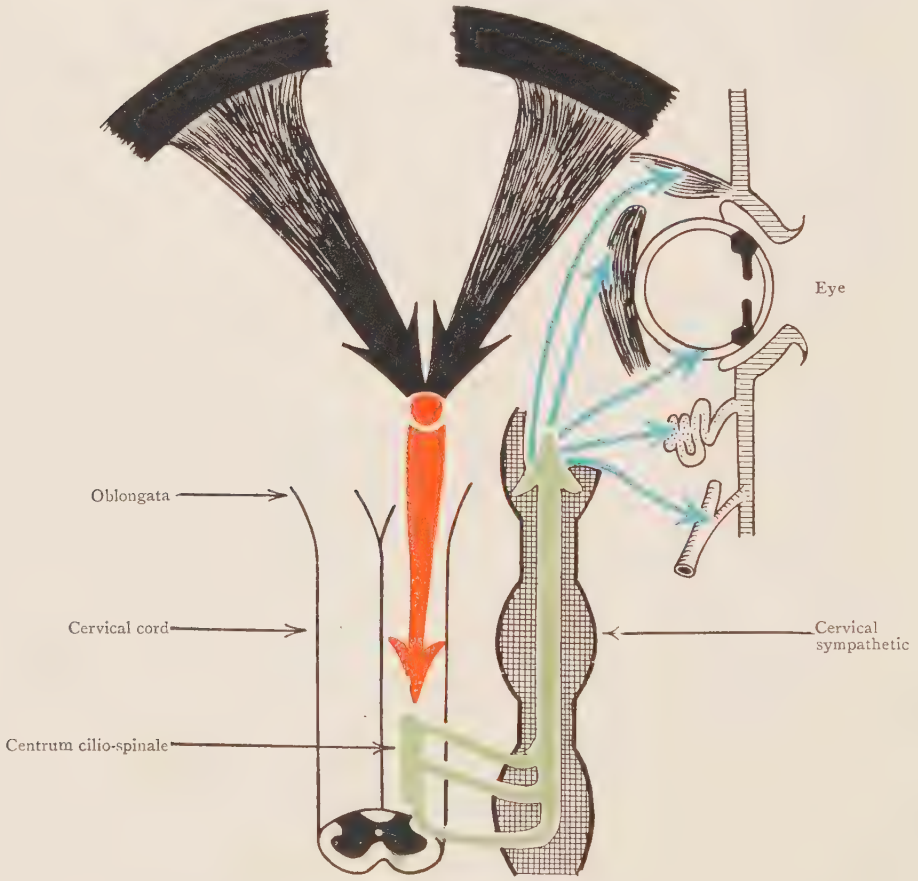


FIG. 31.—DIAGRAM IN EXPLANATION OF HORNER'S (CLAUDE BERNARD'S) SYMPTOM COMPLEX IN LESIONS OF THE UPPER PART OF THE CORD.

phenomena are an accompaniment of those cases in which the roots are damaged on the proximal side of the point at which the rami communicantes are given off.

(δ) By lesions of the rest of the cervical cord, if the bulbo-spinal connecting fibres are divided (*vide* Fig. 31).

Irritation of the centrum cilio-spinale is a rare condition. It may accompany lesions of the dorsal cord immediately below the centrum, and is marked by spasmodic mydriasis (contracture of the dilator pupillæ) and exophthalmos.

(*b*) Disturbances of Respiration.

These are due chiefly to pathological processes affecting the region of the phrenic centre (C_3—C_5). Damage to this centre causes paralysis of the diaphragm—a condition which, when fully developed, leads always to a fatal termination. If the phrenic centre is irritated by a lesion situated in its immediate vicinity, the condition reveals itself by the onset of hiccough, cough, dyspnœa, and vomiting. Lesions situated at a higher level are also accompanied by respiratory disturbances, which are generally grave and indeed eventually fatal. Here, however, we have to do with a condition of impairment of the neighbouring respiratory centres of the medulla (distant effect).

(*c*) Affections of the Pulse.

A characteristic feature of lesions of the upper cervical cord is a (frequently merely transitory) slowing of the pulse. This also is due to the proximity of the medulla, and is to be regarded as a result of irritation of the vagus centre therein situated. More rarely a persistent bradycardia, with occasional attacks of general muscular spasm, is noticed as a symptom of lesions of the upper cervical cord; these phenomena urgently require further investigation and explanation, especially in regard to their relation to the condition known as Adams-Stokes disease. It would appear, however, that the absence of a dissociation between the auricular and ventricular rhythm is characteristic of the "neurogenic" form of persistent bradycardia as distinguished from the "cardiogenic."

(*d*) Vaso-Motor Secretory Disturbances.

Enormous elevation of temperature (109° to 111° F.) is a very frequent accompaniment of injuries to the cervical cord. In rare cases (generally such as exhibit also a slowing of the pulse) we find the temperature, on the contrary, lowered (89° to 86° F.). In the present state of our knowledge we can only regard these disturbances as due to serious impairment of a complicated heat-regulating mechanism in the medulla. We are not at present justified in assuming the existence of special temperature centres in the cord.

Different, again, are the conditions governing the vaso-motor innervation of the face, which is often typically affected in cervical lesions, and whose centres, like those of the above-discussed oculo-pupillary mechanism, we are able to localize. We have here a mechanism whose lowest (most peripheral) centres are in the cervical sympathetic, and in immediate dependence on the nuclear groups of the cervical cord These cell-groups appear to lie at the

same level as the centrum cilio-spinale; they are not, however, to be found in the lateral horn, but probably in the central cell complex of the ventral horn. The connection between the spinal and the sympathetic centres is here also made by the anterior roots of the eighth cervical and first and second dorsal segments and their white rami communicantes.

It would seem that secretory fibres for the sweat glands of the facial integument run in close proximity to the corresponding vaso-motor fibres, for in cases such as we have described above, in which paralytic myosis, enophthalmos, and narrowing of the ocular aperture, are present, we often find, not only vaso-motor paralysis, marked in early cases by heat and redness, and later by coldness of the surface and cyanosis (*vide* p. 22), but also anidrosis of the same regions (Horner's symptom complex). As regards these anomalies of perspiration, our knowledge of the causation of the symptom complex is not as yet exact, and the difficulty of giving a completely satisfactory account of the matter is increased by the fact that in rare cases, instead of the usual anidrosis, we are confronted with a condition of hyperidrosis. This fact makes it, in my opinion, probable that the spinal centre for facial perspiration, though lying very near the centrum cilio-spinale and the spinal vaso-motor centre for the face, is nevertheless yet at a sufficient distance from them to be, in some cases, merely irritated by the proximity of a lesion involving them, while remaining itself uninjured.

(*e*) Special Condition of Reflexes.

In "high" total transverse lesions of the cord—*i.e.*, those affecting the cervical and upper dorsal regions—the reflexes are practically always totally abolished over the paralyzed regions. On pp. 32 and 33 we have considered certain attempts to explain this paradoxical phenomenon.

It goes without saying that in paralytic myosis the inconstant cilio-spinal reflex is likewise absent. It consists of a dilatation of the pupil produced by stimulation of the skin of the neck on the same side.

The foregoing is subject to an important qualification, inasmuch as lesions in the region of the four upper cervical segments generally cause immediate death (respiratory paralysis, interference with the important vital centres in the medulla), so that only in a small proportion of such cases can the symptoms above described develop.

II. DIAGNOSTIC CHARACTERS OF LESIONS AFFECTING THE LOWER PART OF THE SPINAL CORD.

As in lesions of the lowest portion of the cord—the so-called "conus terminalis"—the characteristic clinical symptoms are due to disturbance of the nervous mechanism governing the bladder, the rectum, and the sexual

function, we have reserved for this portion of our work a connected account of the mechanism in question.

(*a*) Innervation of the Bladder.

The spinal centres for the closing and the emptying of the bladder—*i.e.*, for the sphincter vesicæ and the detrusor urinæ—are situated in the grey substance of the third and fourth, perhaps also of the fifth, sacral segments. The motor fibres which originate in these centres pass to the bladder by the pudendal nerve and the inferior hypogastric plexus. In the latter are inserted cell-complexes which, belonging to the collateral ganglion system, represent the peripheral or sympathetic (more strictly speaking, para-sympathetic or autonomous, *vide* p. 20) centres for the bladder functions.

As the spinal centres are superior to the sympathetic, so are they, in their turn, under the influence of cerebral centres. Impulses from those centres, which are partly subcortical in situation (corpus striatum and optic thalamus), and partly cortical, reach the sacral cord probably by way of the antero-lateral columns. Recently the existence of routes for these fibres in the posterior columns also has been maintained by certain investigators. Their views are, however, not as yet generally accepted. We may justifiably conclude that the cerebral impulse has opposite effects on the sphincter and the detrusor respectively, causing relaxation of the former at the same time as it excites contraction in the latter.

Not only centrifugal stimuli, however, flow towards the sacral bladder centre. It receives centripetal stimuli also, and these from the vesical mucous membrane itself. These centripetal fibres, which enter the cord with the posterior roots of the second, third, and fourth sacral segments, form the afferent limb of a reflex arc, by the instrumentality of which, when distension of the bladder reaches a certain degree, evacuation (=sphincter relaxation+ detrusor contraction) is brought about reflexly, without the co-operation—nay, even in spite of the opposing action—of conscious impulses. Sensory fibres, using the term in its narrower sense, are distributed to the bladder wall, as well as the centripetal reflex fibres above alluded to. They pass upwards to the brain, and give us the sensation of urinary pressure and desire to micturate.

(*b*) Innervation of the Rectum.

The conditions here are quite analogous to the foregoing, but somewhat simpler. Here, too, we have a centre in segments: S_3—S_4(—S_5); here, too, efferent fibres in the pudendal nerve and the inferior hypogastric plexus, in which latter also are inserted para-sympathetic ganglia. Here again we have centripetal fibres, some of which pass to the centre, forming the afferent limb of a reflex arc, while others pass upwards to the brain and give the sensation

of rectal pressure and desire for defæcation; here, too, finally, we have efferent fibres in the antero-lateral (or posterior ?) column, which convey voluntary impulses from the brain.

The conditions are simpler than those obtaining in the case of the bladder in the following respects:

1. The efferent fibres last mentioned are of cortical origin only. We are not entitled to affirm the existence of any definite subcortical defæcation centres.

2. The conditions of innervation are less complicated, inasmuch as only a sphincter action (contraction or relaxation) is involved; there is no simultaneous influence on a detrusor in an opposite sense. The function of a detrusor is performed by the abdominal muscles acting voluntarily under the influence of spinal centres situated at a higher level.

(*c*) Innervation of Male Genital Organs.

The capacity for coition depends on the capacity for (1) erection, (2) ejaculation.

Two factors play their part in erection: (*a*) The overfilling of the corpora cavernosa. This is brought about by a relaxation of vascular tone in the arteries of the penis, which causes the flow of an increased volume of blood to that organ. (*b*) This increase of volume is accompanied by an increased firmness and solidity, due to hindrance to the venous outflow, which, again, is due to tonic contraction of the transversus perinei and of the bulbo- and ischio-cavernosus muscles.

In ejaculation, clonic spasms of the two last-named muscles come into play, commencing when, by means of a reflex peristalsis of the ampulla vasorum deferentium, the vesiculæ seminales and the ductus ejaculatorii, the semen has found its way into the membranous portion of the urethra.

The relaxation of arterial tone, which plays so important a part in erection, is due partly to inhibition of vaso-constrictor, partly to stimulation of vaso-dilator centres. These centres are partly spinal (S_1—S_3), partly para-sympathetic, cell-groups of the inferior hypogastric plexus. They are connected, through the lateral column of the cord, with the vaso-motor centres of the medulla and the brain.

The centres controlling the tonic action of the transversus perinei, bulbo- and ischio-cavernosus, alluded to above, are situated in the grey matter of the third and fourth sacral segments, as also are those regulating their clonic action in the act of ejaculation. The supranuclear neurons probably take their course in the lateral columns of the cord.

To the foregoing it may be added that, in addition to the centripetal fibres from the genital integument which give to the genital act somewhat of a reflex

character, the normal functioning of the male sexual apparatus requires also centrifugal tracts which shall convey psychical stimuli.

It follows from these preliminary remarks that disturbances in the innervation of the bladder, rectum, and sexual apparatus, may be produced not only by lesions of the terminal section of the cord, but also (owing to interruption of the supranuclear tracts) by injury or disease of the cord at any level. For this reason we have had to make earlier allusion to these disturbances (p. 34).

We must now state the distinguishing features which enable us to make a differential diagnosis in a case of rectal, vesical, or genital disturbance, and to state whether the lesion causing the disturbance is situated in the nuclear (sacral) or supranuclear region of the cord.

1. Bladder Affections.

A. Lesion above the Centrum Vesico-Spinale.

Abolition of Cerebral (Voluntary) Control over the Bladder and of the Sensation of Urinary Pressure and Desire for Micturition.—The reflex mechanism alone is concerned in the emptying of the bladder. As a rule, therefore, we have a condition of active or intermittent incontinence—"automatism of the bladder" or *incontinence à jet*—in which, as soon as the distension of the bladder reaches a certain degree, afferent impulses from the stretched bladder wall bring about a reflex and involuntary emptying of the viscus by a jet of urine.

Exceptionally—in certain cases of total transverse lesions, for instance—this reflex emptying effect is also abolished. The explanation of these cases is probably the same as was put forward for an analogous condition of tendon reflexes in certain cases of transverse division of the upper cord (*cf.* pp. 32 and 33). In cases such as we are considering we have retention of urine, which, unless help is given by catheterization, may lead to rupture of the overfilled bladder.

B. Lesions in the Region of the Centrum Vesico-Spinale (S_3—S_4).

Permanent Relaxation of both Sphincter and Detrusor.—The clinical picture presented here is one of passive or permanent incontinence—*incontinence vraie.* There is a continuous dribbling of urine in most cases; but if, as is fairly frequently the case, the neck of the bladder possesses a considerable degree of elasticity, it may withstand for a while the pressure of the urine resting upon it. The condition is then one of ischuria paradoxa, or *incontinence par regorgement.* When the engorgement of the bladder reaches a certain point, and not until then, the urine begins to dribble away.

In rare cases this elasticity is very considerable, and may even lead to retention of urine instead of incontinence in any form.

2. Rectal Affections.

A. Lesions above the Centrum Ano-Spinale.

Abolition of Voluntary Control of the Sphincter Ani and of the Desire for Defæcation.—With this the reflex contraction of the sphincter is maintained. There is, in fact, generally some degree of spasm of the sphincter, which makes itself evident on introduction of the finger, and affords clear proof of the supranuclear situation of the lesion. In cases of total transverse lesions the reflex contraction is sometimes abolished.

The clinical picture is that of rectal retention of fæces.

B. Lesions in the Region of the Centrum Ano-Spinale (S_3—S_4).

Permanent Paralysis of the Sphincter (*Incontinentia Alvi*).—The incontinence is complete or incomplete, according to the degree of elasticity in the sphincter. In incomplete incontinence hard fæcal masses may be retained.

3. Affections of the Genital Organs.

A. Lesions situated above the Centrum Genito-Spinale.

Vaso-constrictor paralysis, leading to engorgement of the corpora cavernosa and increase in volume of the penis, is here the most prominent symptom. Actual priapism—*i.e.*, complete and lasting erection—is only occasionally seen, and never in cases of total transverse lesion. Where it is present, a condition of tonic contraction of the transversus perinei, bulbo- and ischio-cavernosus, due to irritation, is to be assumed. Occasionally a condition of hyper-reflex in the ejaculatory apparatus makes itself apparent—a condition in which the most trifling stimuli lead, if not to actual ejaculation, at any rate to clonic contractions of the muscles concerned in the act.

B. Lesions in the Region of the Centrum Genito-Spinale (S_1—S_4).

These throw out of action in most cases the whole mechanism of erection and ejaculation, with complete impotence as a result. Occasionally a condition of incomplete impotence or dissociated disturbance of the genital function is found. In very circumscribed lesions (small hæmorrhages, sclerotic foci, etc.) we may have, for instance, abolition of ejaculatory power and of the orgasm, while capacity for erection remains. Such investigations as have been made up to the present into the pathological anatomy of these cases render it probable that the erection centre is situated somewhat higher than the ejaculation centre (S_1—S_3, S_3—S_4, respectively).

Other Characteristic Features of Lesions of the Conus Terminalis.

Clinically we include under the term "conus terminalis" the three last sacral segments and the coccygeal segment. Pure conus lesions, which are not common, have an extremely characteristic symptomatology.

(*a*) **Passive incontinence, ischuria paradoxa, or retention of urine.**

(*b*) **Incontinence of fæces.**

(*c*) **Impotence, or dissociated disturbance of the genital function.**

Accompanying the foregoing, which we have fully dealt with above, we have:

(*d*) **Peri-ano-genital anæsthesia.**

This occupies an area suggesting that occupied by the leather patch in a pair of riding breeches. The area is made up of the third, fourth, and fifth sacral segment areas (*vide* left side of Fig. 29).

A further characteristic feature is the—

(*e*) **Absence of any impairment of motility or reflex in the muscles of the lower extremity,** owing to the fact that the segment innervation of the great majority of the small muscles of the foot, does not extend below the second sacral; only flexor longus digitorum, flexor hallucis longus and brevis have a portion of their nucleus in the third sacral; the same applies to the Achilles reflex.

The pure conus syndrome is therefore not to be mistaken. It is, however, found pure seldom, and under strictly circumscribed conditions only (intramedullary hæmorrhages, very small gliotic foci, metastases, etc.). Where the conus is damaged or destroyed by extramedullary injuries or morbid processes, the pure symptom complex can only very rarely be developed, inasmuch as the formation of the cauda equina commences from the third lumbar segment, and the conus is entirely enclosed within the cauda. Only in exceptional cases, therefore (of fracture or dislocation of the vertebral column, for instance), can a pure conus lesion be caused—cases, namely, in which the fibres of the cauda equina succeed in escaping injury, and the conus alone suffers, in spite of its central and protected situation. Cases have been already observed in which fractures of the sacrum or of the last lumbar vertebra have caused injury of the centrally situated roots only, and, as these all spring from the lower sacral segments, the pure conus symptom complex has developed.

The peculiar arrangement of the terminal spinal roots in the cauda, due to the disparities in growth between the vertebral canal and its contents (*vide* Fig. 32), is the cause of many difficulties in diagnosis. As in the cauda equina all the sacral and coccygeal and the three last lumbar roots pass downwards together, from the termination of the cord at the level of the first lumbar vertebra until the issue of each at its corresponding vertebral foramen, it is easy to understand that an injury destructive of the cauda equina about the

junction of the third and fourth lumbar vertebræ causes symptoms which are clinically hardly distinguishable from those produced by destruction of the terminal segment of the cord (through fracture of the first lumbar vertebra, for example). It is, in fact, the case that, in disturbances of innervation due to trauma in this region, examination with a view to the discovery of deformities, visible, revealed by X-ray examination, or ascertainable by palpation, and of areas of special sensitiveness to pressure, is of most assistance in forming a diagnosis as to the level of the lesion—a matter of eminent importance from a surgical point of view. It is quite otherwise in the case of morbid processes such as malignant, tuberculous, and syphilitic growths.

The following table should be of some assistance in differential diagnosis in these cases. Even with its aid, however, one must be prepared for many an error in diagnosis.

	LESIONS	
	Of the Lower End of the Cord.	**Of the Cauda Equina.**
1	In some cases (with central foci of disease) "dissociated" disturbance of sensation—temperature sense and sensibility to pain affected, while tactile sensibility is retained	All forms of sensation always affected (*vide* p. 29, posterior roots)
2	Spontaneous pains rare and generally slight	Spontaneous pains (pseudo-neuralgias, anæsthesia dolorosa, root pains, *vide* pp. 30 and 31), especially in the perineum, over the sacrum, in the bladder (generally very severe). In "compression" affections generally precede symptoms of impaired function
3	Fibrillary twitchings in the paralyzed muscles rare	Fibrillary twitchings in the paralyzed muscles (peroneal region) frequent
4	Distribution of symptoms almost always symmetrical	Distribution of symptoms frequently unsymmetrical
5	Marked tendency to sacral bedsore	Somewhat slighter tendency to sacral bedsore

The peculiar features presented by total unilateral lesions in the lowest region of the cord are dealt with on p. 38.

APPENDIX.

Fig. 32 is intended to explain, *inter alia*, the relation of the separate spinal segments and pairs of roots to their corresponding vertebral spines.

Only in the uppermost cervical region is the origin of the spinal roots at the same level as their place of exit from the vertebral canal. The farther one proceeds downwards, the greater is the displacement between the points at which the roots spring from the cord and those at which they pass out of the

spinal canal. There are, of course, individual peculiarities, but in general it may be said that the first dorsal segment is opposite the seventh cervical

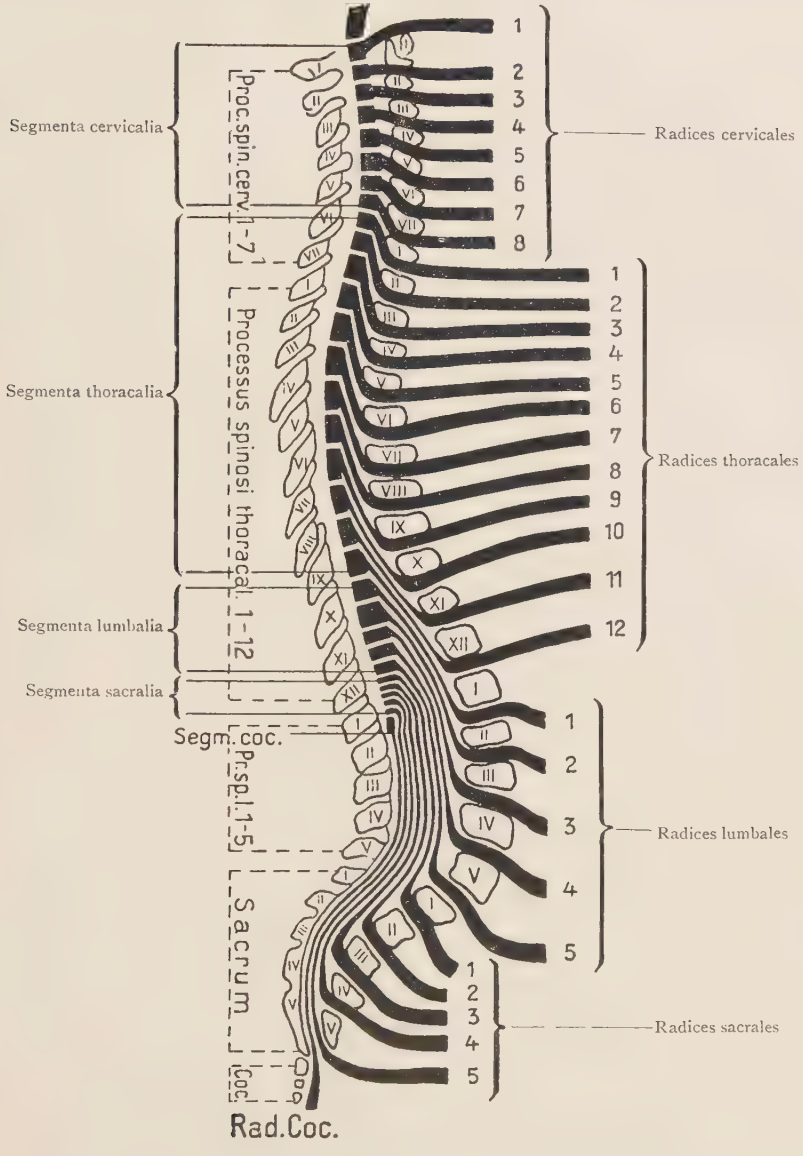


FIG. 32.—TOPOGRAPHICAL CORRELATIONS BETWEEN THE SPINAL SEGMENTS, THE BODIES OF THE VERTEBRÆ, THE SPINOUS PROCESSES, AND THE POINTS OF EXIT OF THE SPINAL ROOTS.

vertebra, the first lumbar segment opposite the tenth dorsal vertebra, and the first sacral segment opposite the first lumbar vertebra. From the second

lumbar vertebra downwards the cord ceases, and only the roots of the cauda equina are found in the spinal canal.

The foregoing statements have reference to the bodies of the vertebræ. As Fig. 32, based on the data of Dejerine and Thomas, shows, the relation to the spines of the vertebræ is in many respects essentially different.

In general, in view of the above conditions and of the overlapping of root fields emphasized on pp. 42 and 43 and in Fig. 15, where operation is undertaken in cases of spinal tumour, laminectomy must be performed 8 to 10 centimetres above the upper border of the anæsthetic zone. This will occasionally be too high—in cases, for instance, of extramedullary tumour, in which, as a result of an arachnitis *adhæsiva circumscripta*, there is, above the tumour, a blocking of the cerebro-spinal fluid which itself causes compression of the cord.

DIVISION II

REGIONAL DIAGNOSIS OF CEREBRAL LESIONS

DIVISION II

REGIONAL DIAGNOSIS OF CEREBRAL LESIONS

A. LESIONS IN THE REGION OF THE BRAIN-STEM.

THE structures which the neuro-pathologist groups under the titles "cerebral trunk," "cerebral axis," or "brain-stem"—*i.e.*, the medulla oblongata, pons, cerebral peduncles, and corpora quadrigemina—reveal their anatomical and physiological interrelationship in the fact that they contain the nuclear areas of origin of all the cerebral nerves except the olfactory and the optic. We are fully justified, too, in placing the two first pairs of nerves in a different category, for they are not to be regarded as peripheral nerves analogous to the ten other pairs, but as portions of the brain which, during the embryonal period, have protruded themselves in a cylindrical or tubelike form.

This anatomico-physiological characteristic of the brain-stem determines also the clinical character of its lesions and their semeiology, for disturbances of function in the areas of origin of the cranial nerves, from the oculo-motor to the hypoglossal, are the significant features of all affections of the brain-stem. Nay, further, only by careful consideration of these signs, of their method and order of appearance, and of their combinations, can we succeed in localizing correctly pathological processes within that region.

A knowledge of the morphology of the brain-stem and of the points at which its nerves spring from it is assumed. Figs. 33 to 35 may serve for recapitulation. Some detailed description, however, of the internal anatomy of these structures cannot be dispensed with, and, in view of what has been said concerning the chief diagnostic feature of affections involving this region, it is essential to acquire an accurate knowledge of the topography of the origins and terminations of the cranial nerves, from the third to the twelfth, included within it. In addition, the course of the chief motor and sensory tracts with which acquaintance has been made in Division I. has to be borne in mind. We may, however, pass over a mass of structural details which are, for the moment, of no diagnostic significance. Thanks to this limitation, the necessary study of the histology of the brain-stem (Chapter I.) becomes comparatively simple, and correspondingly so the deduction of general rules for the localization of injuries and diseases affecting it (Chapter II., Part I.). The description

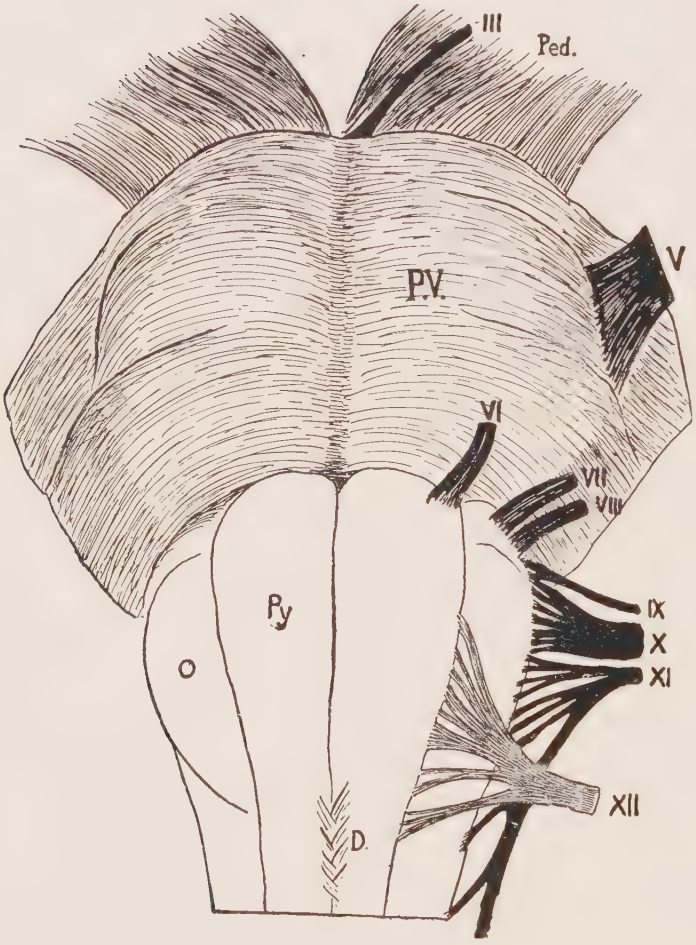


FIG. 33.—VENTRAL ASPECT OF THE BRAIN-STEM, SHOWING POINTS OF EMERGENCE OF CRANIAL NERVES.

Py. = Pyramis medullæ oblongatæ.
D. = Decussatio pyramidum.
O. = Oliva.
P.V. = Pons Varolii.
Ped. = Pedunculus cerebri.
XII. = Nervus hypoglossus.
XI. = Nervus accessorius Willisii.
X. = Nervus vagus.
IX. = Nervus glosso-pharyngeus.
VIII. = Nervus acusticus.
VII. = Nervus facialis.
VI. = Nervus abducens.
V. = Nervus trigeminus.
III. = Nervus oculo-motorius.

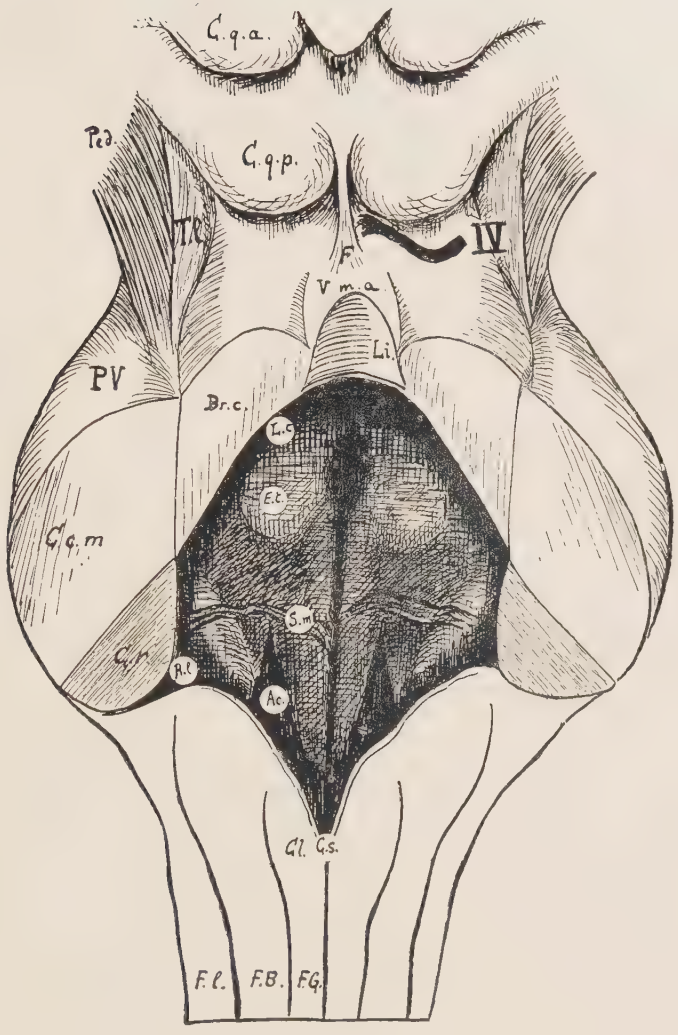


FIG. 34.—DORSAL ASPECT OF THE BRAIN-STEM, WITH FOURTH VENTRICLE LAID OPEN.

F.G. = Funiculus Golli.
F.B. = Funiculus Burdachi.
F.l. = Funiculus lateralis.
Cl. = Clava medullæ oblongatæ.
C.s. = Calamus scriptorius.
A.c. = Ala cinerea.
R.l. = Recessus lateralis fossæ rhomboideæ.
S.m. = Striæ medullares.
E.t. = Eminentia teres.
L.c. = Locus cœruleus.
C.r. = Corpus restiforme s. crus cerebelli post.
C.c.m. = Crus cerebelli med. s. brachium pontis.
Br.c. = Brachium conjunctivum s. crus cerebelli anterior.
Li. = Lingula cerebelli.
V.m.a. = Velum medullare anterius.
P.V. = Pons Varolii.
T.l. = Trigonum laquei s. lemnisci.
Ped. = Pedunculus cerebri.
C.q.p. = Corpus quadrigeminum post.
C.q.a. = Corpus quadrigeminum ant.
F. = Frenulum veli medullaris ant.
IV. = Nervus trochlearis.

of certain more complicated anatomical conditions relating to the course, distribution, and inter-relationships of nerve fibres, a knowledge of which is

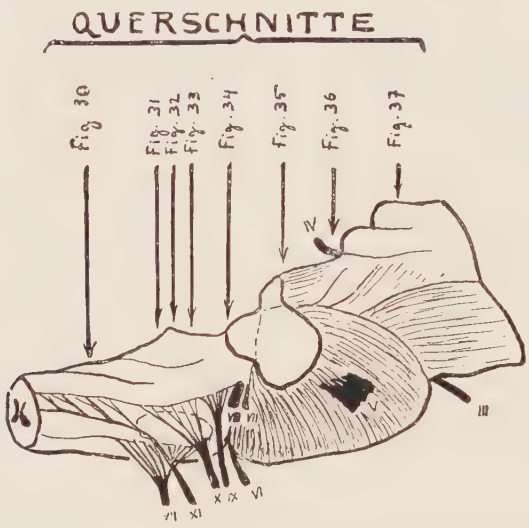


FIG. 35.—LATERAL ASPECT OF THE BRAIN-STEM.

The figure shows the levels at which the transverse sections (Querschnitte) shown in Figs. 36 to 44 are taken.

necessary for the due appreciation, from the point of view of regional diagnosis, of certain special symptom complexes, will, together with a consideration of the latter, be reserved for the third chapter.

CHAPTER I.

STRUCTURE OF THE BRAIN-STEM.

OUR illustrations (Figs. 36-44) are so arranged that the left half illustrates a transverse section at a particular level of the medulla, pons, or cerebral peduncle. The level at which the section is made is seen in Fig. 35. On the right half is a diagrammatic representation of those structures only which are of importance for our discussion of regional diagnosis in the ensuing chapter—the origins, terminations, and intracentral tracts of the cranial nerves, and the great motor and sensory tracts for the body generally. Progressing in a frontal direction from the cervical cord, we shall describe each transverse section in turn. In this manner the more complicated conditions are led up to naturally, and elucidated by the more simple.

1. Section through the Upper Cervical Cord (C_2) (Fig. 36).

The illustration represents the topographical anatomy of the grey matter and of the white tracts, as fully described in Division I.

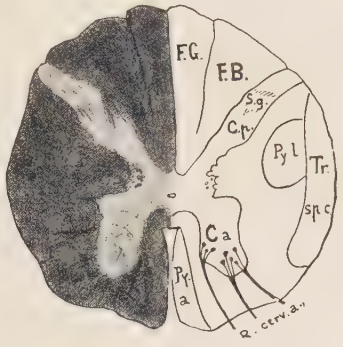


Fig. 36.

F.G.=Funiculus Golli; F.B.=Funiculus Burdachi; S.g.=Substantia gelatinosa Rolandi; C.p.=Cornu posterius; C.a.=Cornu anterius; R.cerv.a.=Radices cervicales anteriores; Py.l.=Fasciculus pyramidalis lateralis; Py.a.=Fasciculus pyramidalis anterior; Tr.sp.c.=Tractus spino-cerebellares.

2. Section through the Lower Portion of the Medulla Oblongata (Level of the Pyramid Decussation) (Fig. 37).

The general structural arrangement remains as in the cord. The transverse section of the grey matter, however, as is seen by a comparison with Fig. 36, gives evidence of a considerable change. Especially noteworthy is it that the

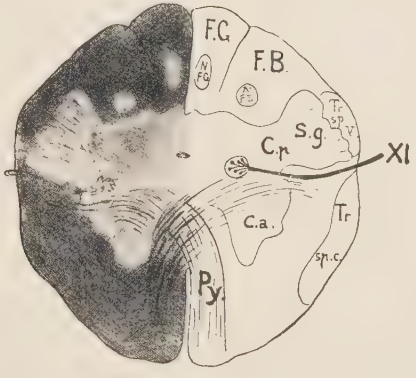


Fig. 37.

N.F.G.=Nucleus funiculi Golli; N.F.B.=Nucleus funiculi Burdachi; Py.=Fasciculus pyramidalis; Tr.sp.V.=Tractus spinalis trigemini; XI.=Nervus accessorius.

dorsal horns have lost their connection with the periphery of the cord, inasmuch as there is no longer any entry of posterior roots. The grey matter is, conse-

quently, terminated dorsally by the substantia gelatinosa, which is here markedly developed. Fibres enter its ganglion cells which come from a considerably higher level—namely, from the pons—and belong to the trigeminus. These fibres form the descending or spinal root of the fifth nerve, so called because its fibres extend downwards to the upper end of the cord. This spinal root is situated, somewhat like a cap, on the external and posterior aspect of the substantia gelatinosa, which may be regarded as its nucleus.

We miss the anterior roots also. On the other hand, a root of the spinal accessory nerve—the motor nerve for the sterno-mastoid and trapezius muscles —issues from a group of cells at the base of the ventral horn. The point of exit of this root is not in the line either of the anterior or the posterior roots, but directly to the side of the medulla.

The situation and topography of the spino-cerebellar tracts are the same as in the cord. So also with the posterior columns. Within these, however, we have at this level two cell complexes—the nuclei of the column of Goll and of the column of Burdach respectively. The fibres of the posterior columns—*i.e.*, the long neurons of the posterior root system described on pp. 7 and 8—find their termination in these nuclei, forming a connection there with sensory neurons of the second order (*vide* p. 9). These, the bulbo-thalamic tracts, are the axis cylinders of the *nucleus funiculi Golli* and the *nucleus funiculi Burdachi*. They are depicted in the next figure.

The most characteristic feature of this section (Fig. 37) is, however, the decussation of the pyramids. From the area on either side of the anterior median fissure, which, in the upper part of the medulla oblongata, contains the whole of the fibres of the cortico-spinal tracts, the majority of those fibres cross over to the opposite lateral column, and descend therein as the lateral pyramid tract. Only a small portion of the fibres remains uncrossed, and maintains its position close to the ventral median fissure as the ventral or direct pyramid tract.

3. **Section through the Medulla Oblongata at the Level of the Posterior Hypoglossal Roots** (Fig. 38).

The grey substance has here undergone still more significant changes. The remnant of the dorsal horn (substantia gelatinosa) is pushed far to the side by the much-enlarged nuclei of Goll and Burdach; its relations to the spinal root of the fifth nerve, however, remain the same as at a lower level. From the nuclei of the posterior column the bulbo-thalamic tracts pass across in a wide sweep to the other side of the medulla, to occupy there the area known as the "laqueus," "lemniscus," or "fillet" (*sensory decussation, decussation of the fillet*).

The ventral horns no longer exist as such. They have resolved themselves into separate cell-groups. The nucleus of the accessory nerve is situated as in

Fig. 37, and from the nucleus of the hypoglossal nerve issue the roots for the motor nerves of the tongue, their course and arrangement closely analogous to those of the spinal anterior roots.

Other structures of the grey matter are the olive and the accessory olive, about which we shall have something to say later.

The pyramids form large compact masses of fibres on either side of the median fissure. The spino-cerebellar tracts, traversed by the accessory nerve, occupy their old position at the periphery of the lateral columns.

Instead of the dorsal median sulcus, a median fissure has now been formed, which penetrates almost to the central canal. With the junction of the median

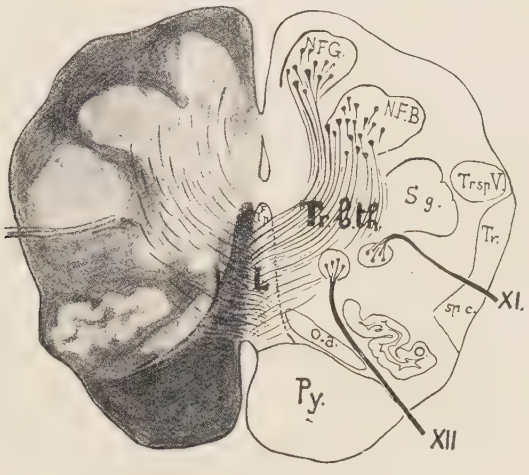


Fig. 38.

Tr.b.th.=Tractus bulbo-thalamici; L.=Laqueus s. lemniscus; F.l.p.=Fasciculus longitudinalis posterior; O.=Oliva; Oa.=Oliva accessoria; XII.=Nervus hypoglossus.

fissure and the central canal—*i.e.*, with the formation of the so-called *calamus scriptorius*—the fourth ventricle takes its commencement.

Behind the area of the fillet is seen the *dorsal longitudinal bundle*. It is not until this level is reached that it forms a sharply defined area in transverse section. Its fibres, however, reach far down into the cord. It is a collection of "association" fibres connecting cerebral with spinal nuclei. On account of its close relations with the nuclei of the ocular muscles and the vestibular system (*vide infra*), it is generally supposed that the dorsal longitudinal bundle is chiefly engaged in maintaining muscular *synergia*—the co-ordination and co-operation necessary for a correct appreciation of our position in space.

4. Section through the Medulla Oblongata (Lower Third of the Fourth Ventricle, Origin of Vagus (Fig. 39).

The fourth ventricle has opened out. Immediately beneath its floor lies the nucleus of the hypoglossal nerve, which in Fig. 38 lay much farther forward —in an axial continuation, in fact, of the ventral horns. The sensory nuclei are, of course, still farther back. Passing from without inwards, we meet—(1) the nucleus of Burdach, here considerably enlarged; (2) the nucleus of Goll, here very small—having already given most of its fibres to the formation of the fillet; (3) the sensory nucleus of the vagus, which is seen in macroscopic

Fig. 39.

Tr.sp.c.d. = Tractus spino-cerebellaris dorsalis; Tr.sp.c.v. = Tract. spino-cereb.ventr.; X. = Nervus vagus; N.a. = Nucleus ambiguus.

preparations through the floor of the ventricle, and gives to the region known as the *ala cinerea* its grey colour.

The motor nucleus of the vagus (nucleus ambiguus vagi) is situated ventro-laterally to the nucleus of the hypoglossal, in the middle of the area occupied by the curving fibres of the fillet decussation. The vagus trunk, made up of sensory and motor fibres for larynx, stomach, œsophagus, heart, and lungs, passes out laterally from the medulla between the tract of Gowers and the lateral cerebellar tract. While the former has kept its old position (dorso-lateral to the olive), the latter (tractus spino-cerebellaris dorsalis), moving in a dorsal direction, has already arrived within the near neighbourhood of the nucleus of Burdach, pushing itself between the periphery of the medulla and the area of the descending root of the fifth nerve. The nucleus of the latter—the substantia gelatinosa Rolandi—lies, as in Fig 8, between it and the

region of the bulbo-thalamic tracts. It is now, however, traversed by the neuron complex of the vagus.

As regards the dorsal longitudinal bundle, fillet, pyramids, hypoglossal roots, and olive, their positions are not markedly changed from those given in the foregoing transverse section (Fig. 38). In addition to the median, we have now a dorsal accessory olive.

5. Section through the Medulla at the Broadest Part of the Fourth Ventricle; Origin of Glosso-Pharyngeal (Fig. 40).

The section has, in its dorsal portion, undergone a considerable broadening, in consequence of the flattening and opening out of the fourth ventricle. The nuclei of the dorsal funiculi, too, now lie beneath the floor of the ventricle, and far to the side, in the so-called *recessus lateralis*. No separation can now

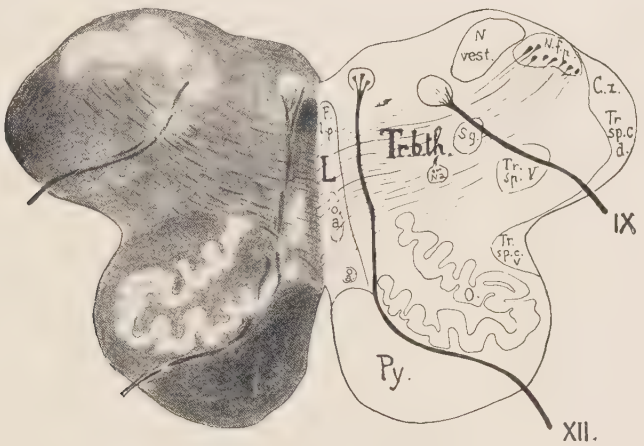


Fig. 40.

N.vest.=Nucleus vestibularis; IX.=Nervus glosso-pharyngeus; C.r.=Corpus restiforme; N.f.p.=Nucleus funiculorum posteriorum.

be made between the nuclei of Goll and of Burdach. We still see their last sparse neurons passing across to the opposite fillet; in the succeeding sections we shall not meet the dorsal nuclei again.

Immediately to the median side of the dorsal nuclei there emerges, at this level, the nucleus of the vestibular nerve (*vide infra*). The hypoglossal nucleus and the motor nucleus of the vagus are seen in the same relative positions as in the more caudal sections. In the place of the sensory nucleus of the vagus appears the glosso-pharyngeal nucleus, in which the gustatory fibres from the posterior third of the tongue and the sensory neurons from the pharynx and the

tympanic cavity find their termination. The glosso-pharyngeal nerve pierces the periphery at a point closely corresponding to that at which the vagus passes through it in Fig. 39. It, too, traverses the substantia gelatinosa, which is here considerably smaller in section, and has withdrawn itself inwards and upwards from the descending tract of the fifth nerve; while the latter has moved still farther from the periphery in a dorso-median direction. The tract of Gowers still occupies its old position; the dorsal spino-cerebellar tract, or direct cerebellar tract of Flechsig, merges dorsally in the restiform body, which, as the inferior peduncle of the cerebellum, connects the latter with the medulla. The dorsal longitudinal bundle, the pyramids, and the fillet have not altered their relative positions. The inner or dorsal accessory olivary nuclei are now enclosed in the substance of the fillet. The olives are here at their greatest development.

6. Section through the Caudal End of the Pons; Origin of Auditory, Facial, and Abducens Nerves (Fig. 41).

In this section the pyramids are "caught" just before their splitting up into separate bundles among the transverse fibres of the pons. Above the pyramid is the transverse section of the upper end of the olive.

FIG. 41.

N.vest.=Nucleus vestibularis; N.coch.=Nucleus cochlearis; N.D.=Nucleus Deitersi; VIII.=Nervus acusticus; VII.=Nervus facialis; VI.=Nervus abducens.

In the dorsal half of the section the area bordering the median line, or raphe, is occupied by the fillet and dorsal longitudinal bundle.

Towards the sides there is a continuous passing over of fibres from the brain-stem into the cerebellum. Here, in the restiform body, the neurons of

the dorsal spino-cerebellar tract are enclosed. The ventral portion, or tract of Gowers, is still situated at the periphery, dorso-lateral to the olive. Its wedge-shaped transverse section exhibits, as in some of the previous sections, a point directed inwards. The substantia gelatinosa and descending tract of the trigeminus show little change from Fig. 40.

As in the latter figure also, the vestibular nucleus lies, close to the surface, beneath the floor of the lateral portion of the fourth ventricle. We now see the fibres of the vestibular nerve, the portion of the eighth which serves to maintain our sense of our position in space, passing between the restiform body and the descending tract of the fifth nerve to the vestibular nucleus. The actual auditory nerve, or nervus cochlearis, has its nucleus in a rounded projection on the lateral and dorsal aspects of the restiform body (the *acoustic tubercle*).

Not superficially under the floor of the fourth ventricle, but deep in the substance of the *formatio reticularis* of the pons, lies the motor nucleus of the seventh or facial nerve. The intracentral course of this nerve is a peculiar one. It passes inwards and backwards towards the floor of the fourth ventricle; when close under the latter it winds itself round the superficially situated abducens nucleus, and turns back through the substance of the pons, emerging in the immediate neighbourhood of the eighth nerve at the cerebello-pontine angle. As the second half of the loop thus formed does not run in the same transverse section as the first, but at a somewhat higher level, we have represented it by a dotted line. A little higher still emerges the sixth nerve or abducens, which supplies the external rectus muscle of the eye. Its roots also are, for the same reason, represented by a dotted line. They emerge from the base of the brain at the lower border of the pons, considerably nearer the middle line than those of the facial nerve. The portion of the facial root which circles round the nucleus of the abducens—the so-called "genu" or "knee" of the facial root—forms the prominence known as the *eminentia teres* on the floor of the fourth ventricle.

Near the vestibular nucleus, at the boundary of the cerebellum, lies the nucleus of Deiters, which, it should be remembered, is the origin of the vestibulo-spinal tract. In order not to complicate the figure, we have omitted the connections between the nucleus of Deiters and the dorsal longitudinal bundle, between the former and the vestibular nucleus, and between the vestibular nucleus and the dorsal longitudinal bundle. These connections are, however, physiologically of great importance, as already stated (pp. 81 and 82: Co-operation of Ocular and Labyrinthine Apparatus in Maintaining Orientation in Space).

7. Section through the Middle Third of the Pons at the Level of the Point of Exit of the Fifth Nerve (Fig. 42).

The ventral portion of the pons (pars basilaris pontis) shows, embedded in the mass of its transverse fibres (which are for the most part connected with the cerebellum by the middle cerebellar peduncle), the pyramid tracts cut across. They do not now form a compact, single bundle, as in the foregoing sections, but are split up into separate fasciculi.

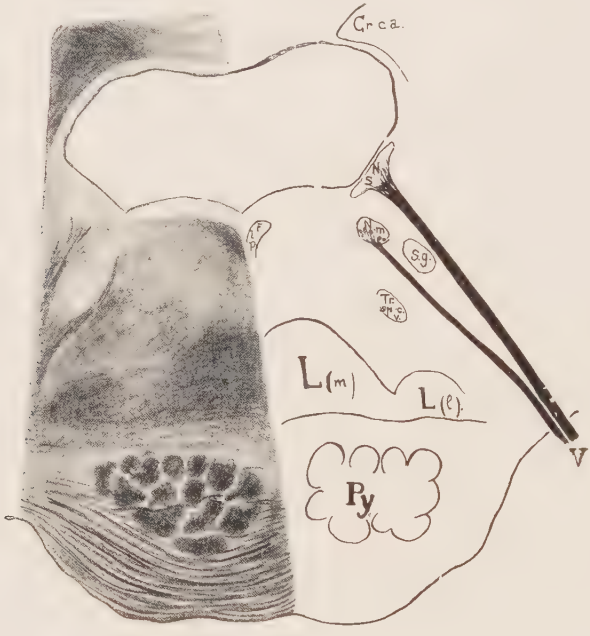


Fig. 42.

L.l.=Laqueus lateralis; L.m.=Laqueus medialis; Ns.=Nucleus sensibilis; N.m.=Nucleus motorius; V=.Nervus trigeminus; Cr.c.a.=Crus cerebelli anterius.

In the dorsal portion of the pons, or the tegmentum pontis, the dorsal longitudinal bundle is seen in its accustomed position. The area of the fillet has now lost its narrow, elongated form and become broader, and at the same time distinctly shorter from back to front, so that the bulbo-thalamic tracts and the dorsal longitudinal bundle are no longer in immediate contiguity. The former, in this and the succeeding sections, are no longer synonymous, without qualification, with the *fillet*. They form now the *median fillet*, while more laterally placed is the *lateral fillet*, an area occupied by neurons of the central auditory tract in their passage from the auditory centres in the brain-stem to the mid-brain.

The nuclear conditions are at this level much simplified. The trigeminus has a motor nucleus which lies in the same sagittal plane as the nucleus of the facial, but some distance from it, in a dorsal direction. From it arises the smaller, motor portion of the trigeminus. It issues from the base of the brain in company with the larger sensory portion comprising the sensory nerves of the face. Their ganglion cells lie, as is well known, in the Gasserian semilunar ganglion; their axis cylinders pass to the sensory nucleus of the trigeminus, which lies at the side of the fourth ventricle, beneath its floor, through which it glimmers with a bluish appearance, which has gained for the area concerned the name of *locus cæruleus*. For the last time we meet with the substantia gelatinosa Rolandi, into which trigeminus fibres continue to penetrate right down to the cervical cord. These fibres come from the descending tract of the trigeminus, which is made up of fibres from the sensory portion of the fifth, which, instead of entering the sensory nucleus, turn in a caudal direction, and traverse the brain-stem for a greater or less distance.

The bundle of Gowers has left its peripheral situation at the cerebello-pontine angle to reach the dorsal portion of the tegmentum at the anterior border of the pons. Our section finds it about half-way through its journey.

8. **Section through the Anterior Third of the Pons; Exit of the Trochlear Nerve at the Velum Medullare Anterius** (Fig. 43).

The basilar portion of the pons has increased greatly in extent. The pyramid bundles embedded in it are still more scattered than in Fig. 42. On

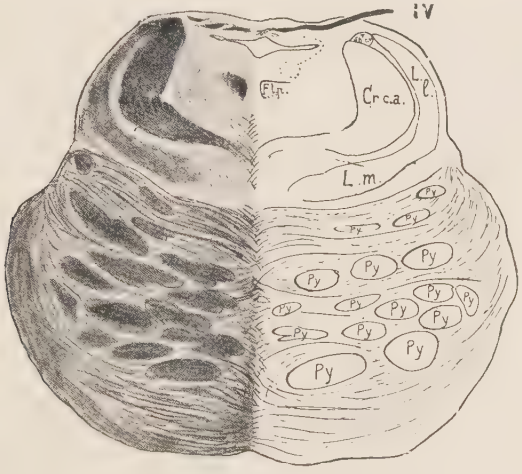


Fig. 43.
IV. = Nervus trochlearis.

the other hand, the volume of the dorsal portion or tegmentum has much diminished simultaneously with the marked contraction in size which the

fourth ventricle has undergone beneath the superior medullary velum. The tegmentum still encloses in its substance the dorsal longitudinal bundle and the fillets (median and lateral). The former still lies close to the median line, and near the floor of the ventricle; the median fillet is at the deepest part of the tegmentum, but has moved somewhat in a lateral direction (the bulbo-thalamic tracts are now leaving each other, and passing in the direction of the optic thalami). The lateral fillet has moved close up to the lateral periphery and velum medullare, and is about to pass into the inferior corpora quadrigemina.

The whole fillet area surrounds like a crescent that occupied by the fibres which, with the superior medullary velum between them, pass from the cerebellum to the corpora quadrigemina. (In Fig. 42 these fibres are shown as they emerge from the cerebellum.) These fibres constitute the superior cerebellar peduncle or brachium conjunctivum. The bundle of Gowers has attached itself closely to this structure, and passes along it, coursing backwards, therefore, to the *vermis cerebelli*.

Within the superior medullary velum we see the decussation of the trochlear nerves. The trochlear—the nerve which supplies the superior oblique muscle of the eye—is distinguished from all other cranial nerves by its dorsal emergence. Some investigators have put forward a phylogenetic explanation of this remarkable anatomical peculiarity of the trochlear, and suggest that it originally supplied a muscle of the "parietal organ."

The nucleus of the trochlear nerve is not seen in this section. It lies farther forward at the proximal extremity of the pons, beneath the inferior corpora quadrigemina and behind the dorsal longitudinal bundle. The manner in which it sends its fibres round the ventricle, to decussate with its fellow in the superior medullary velum, is indicated by a dotted line.

9. **Section through the Cerebral Peduncles and the Superior Corpora Quadrigemina (Exit of Oculo-Motor Nerve)** (Fig. 44).

In this section also we distinguish a pars basilaris (crusta) and a tegmentum. The boundary between the two is formed by the substantia nigra of Sömmering. In the pars basilaris, or crusta, run the pyramids, closed in on either side by tracts of fibres which connect the cerebral cortex and the pons—mesially by the occipito-temporo-pontine, laterally by the fronto-pontine tract.

In the tegmentum we see, immediately beneath the ventricle, which has here narrowed and become the aqueduct of Sylvius, the oculo-motor nucleus (nucleus of third nerve). We have represented the latter in a manner which does not fully portray the somewhat complicated anatomical conditions it presents. We shall have to deal with these later, but may confine ourselves here to the coarser anatomy of the parts. In its course to the base of

the brain, where it emerges at the median border of the crusta, the oculo-motor nerve traverses the red nucleus of the tegmentum, whose importance as the point of origin of Monakow's bundle has been already alluded to (*vide* p. 4).

The oculo-motor nucleus, whose neurons supply the sphincter iridis, the ciliaris muscle, and all the external muscles of the eye, except the external

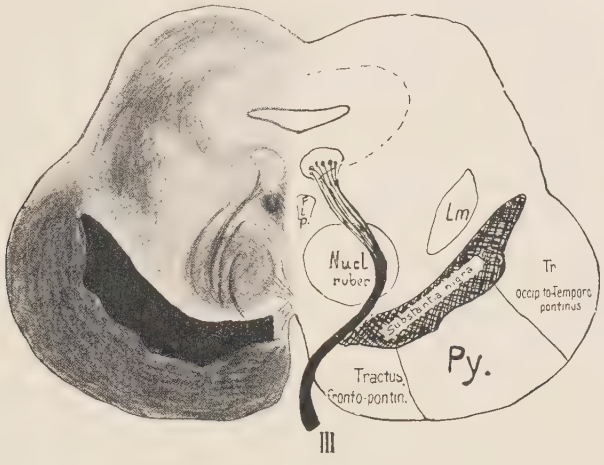


FIG. 44.
III.=Nervus oculo-motorius.

rectus and superior oblique, has the closest relations with the dorsal longitudinal bundle. The median fillet has moved still farther laterally than in Fig. 43. We are now only a few millimetres below (caudal to) the point at which the mass of the bulbo-thalamic tracts bury themselves in their goal, the optic thalamus.

CHAPTER II.

I. GENERAL RULES FOR THE LOCALIZATION OF INJURIES AND DISEASES OF THE BRAIN-STEM.

WE must endeavour in the first place to deduce, from what has been said in the foregoing chapter concerning the anatomy of the brain-stem, the structural principles which govern its formation, and have their correlative in principles of localization of similar general applicability.

(*a*) The nuclei of the cranial nerves are almost all situated in the dorsal part of the brain-stem. We may distinguish three layers, of which the first two occupy the tegmentum of the pons and cerebral peduncles. Most dorsally situated—*i.e.*, close beneath the floor of the fourth ventricle or the aqueduct

of Sylvius—lie the nuclei of the hypoglossal, vagus (sensory), glosso-pharyngeal, vestibular, abducens, trigeminus (sensory), trochlear, and oculo-motor nerves. In a more ventrally situated layer are the nuclei of the accessory and facial nerves and the motor nuclei of the vagus and trigeminus. This layer is situated in the "formatio reticularis." A still more ventral situation is occupied by the cochlear nerve alone, in the "acoustic tubercle."

(*b*) The fibre complexes of the cranial nerves pass from their nuclei above mentioned to the base of the brain; the majority follow a direct course, but the route followed by the vagus (motor) is somewhat circuitous, and that of the facial still more so. The trochlear nerve (fourth) is alone in emerging dorsally (at the superior medullary velum); it is exceptional in another respect also—namely, that, after leaving their nucleus, its fibres undergo a complete decussation with their fellows of the opposite side. We shall have something to say later concerning a partial decussation of the oculo-motor fibres to which allusion has not yet been made. All the other nerve roots of the brain-stem, both motor and sensory, emerge uncrossed—*i.e.*, on the same side as their nuclei.

(*c*) Ventral, or rather ventro-mesial, to the region of the cranial nerve nuclei—*i.e.*, the floor of the fourth ventricle and the formatio reticularis—lies the area occupied by the sensory tracts for the trunk and extremities. We know that this is termed the "fillet" (*laqueus, lemniscus*), and we have seen that the greater part of this structure is made up of the bulbo-thalamic tracts, which form connecting neurons for the dorsal columns—*i.e.*, for the long fibres of the posterior spinal root system (*vide* pp. 6-8). They form afferent channels, therefore, for deep sensibility and the tactile sense (*vide* pp. 15 and 16). We became acquainted also in Division I. with short fibres of the posterior root system, which terminate in the dorsal horn, and to which the spino-thalamic tracts connect themselves. These spino-thalamic tracts, which decussate soon after their origin, pass without further decussation to the optic thalami, and mingle with the bulbo-thalamic tracts after the fillet has been constituted by the latter. They convey to our consciousness all painful and thermal, and in addition some tactile, impressions. According to the present state of our anatomico-pathological knowledge on the point, it would appear that, in the portion of the fillet nearest to the raphe or middle line, only bulbo-thalamic fibres are contained, so that, in lesions affecting exclusively the most mesial portion of the fillet, the chief symptoms are disturbances of the muscular sense in the trunk and extremities. Only when the lesion affects also the lateral part of the fillet is it usual to find sensibility to pain and to thermal and tactile impressions affected. The circumstance that these may be affected also in lesions of the formatio reticularis (*vide* p. 92) gives support to the view expressed by several authors, that some portion of the spino-thalamic tract must also pass through this region on its way to the thalamus. While, in the caudal

portion of the brain-stem, the fillet areas of the two sides are in contact, from the mid-pons region they separate farther and farther from each other.

(*d*) The pyramids occupy the most ventral region of the medulla and the cerebral peduncle. Within the pons their situation is less superficial. Here also they are split up into numerous separate fasciculi by the interposition of the transverse fibres of the pons. For them, as for the sensory tracts for the trunk and extremities, the statement holds that, while in close apposition to one another in the more caudal portion of the brain-stem, they diverge from each other more and more as one proceeds in a frontal direction. This divergence begins, in fact, in the medulla.

(*e*) At their passage from the internal capsule of the brain into the brain-stem (cerebral peduncle), the pyramids comprise, besides the central motor tracts for the trunk and extremities, those also for the muscles of deglutition and the facial, laryngeal, and lingual muscles. At this level the expressions "pyramid tracts" and "cortico-spinal tracts" are by no means co-extensive, for the pyramids include the cortico-bulbar tracts also—*i.e.*, the supranuclear tracts for the trigeminus (motor), facial, vagus (motor), and hypoglossal. These components, however, detach themselves successively from the united "complex" during its passage through the brain-stem, to connect, after crossing the middle line, with their respective nuclei.

(*f*) Longitudinal fibre tracts encountered throughout the greater extent of the brain-stem are—dorsally, near the middle line, the dorsal longitudinal bundle; laterally, the descending or spinal root of the trigeminus, with its nuclear column, the substantia gelatinosa Rolandi. In the medulla we have, at the lateral periphery, the united spino-cerebellar tract; while in the pons only its ventral portion (tract of Gowers) is seen, following the course shown in Figs. 42 and 43.

If the foregoing structural features are kept in mind, there will be no difficulty in understanding the regional diagnostic principles which we here base upon them.

Owing to the small calibre of the medulla, very small lesions, affecting especially its more caudal regions, may give rise to very marked symptoms, and especially to symmetrical affections in which both sides of the body are involved. Thus, a minute hæmorrhage or area of softening may cause complete paralysis of the tongue owing to the very slight distance which separates the hypoglossal nucleus on one side of the medulla from that on the other. As the brain-stem broadens steadily in a frontal direction, the conditions necessary for the production of bilateral affections are far less often present in the pons and the tegmentum than in the medulla. The close apposition to each other of the right and left fillet area, however, explains the fact that in the caudal half of the pons bilateral affections in the fillet region, leading to bilateral anæsthesia of the trunk and extremities, are fairly frequently produced by

small lesions. In small mesial lesions in the pons, the anæsthesia as a rule only extends to the deep sensibility (muscular sense).* The detailed account of the topography of the sensory tracts in the pons given on pp. 90 and 91 furnishes an explanation of this fact, as also of the fact that the more laterally the lesion is situated in the fillet area, thus encroaching on the formatio reticularis, the more likely are the temperature sense and sensibility to pain to be affected. It would appear, too, that the tracts conveying painful sensations are more medially situated than those conveying sensations of temperature. The sensory disturbances produced by unilateral interruption in the fillet tract are, of course, contralateral, and take the form chiefly of crossed hemiataxia, unless the interruption has taken place in the most caudal part of the medulla, distal to the " decussation of the fillet."

Homolateral hemiataxia (of cerebellar type, *vide* pp. 29 and 30) is caused by unilateral affections of the spino-cerebellar tract or of the corpus restiforme. The subject will be further dealt with in the section on the Cerebellum.

In the *formatio reticularis*, in close proximity to the tracts for temperature sense, there must run fibres having relation to vaso-motility, for in lesions in this neighbourhood contralateral hyperidrosis is observed, as also contralateral pallor and coldness of the skin, with lowering of its temperature by 1° or 2° (ponto-bulbar, vaso- and thermo-asymmetry). The pyramids are in such close apposition to each other in the medulla that minimal lesions may cause a condition of tetraplegia. That this is not more frequently observed is due to the fact that lesions of the oblongata are, owing to the inclusion of many important vital centres in the vagal nucleus, generally fatal. A very rare condition is that of *hemiplegia cruciata*, which is produced by lesions situated laterally at the pyramid decussation in such a position that the tracts for the arm are interrupted before, those for the leg after, they have passed to the opposite side. We have, then, a contralateral arm and a homolateral leg paralysis of central cortico-spinal type (*vide* Fig. 45).

Fig. 45.—Mode of Origin of Crossed Hemiplegia.

+ = Paralyzed.

In the basilar portion of the pons, in which the pyramids are split into separate bundles by the numerous transverse pontine fibres, small lesions may

* There is a certain amount of evidence pointing to the conclusion that in the fillet area the fibres corresponding to the sensory apparatus of the sacral cord have the most dorsal situation, close to the posterior longitudinal bundle, and that passing in a ventral direction we meet, in order, the lumbar, dorsal, and cervical fibres, the latter in close proximity to the pyramid tracts.

fail entirely to produce symptoms, either because too few pyramid fibres are affected, or because the pyramids have escaped altogether.

If, however, we keep in mind the case of a unilateral pyramid lesion (found most frequently in lesions of the brain-stem), it is clear that an interruption of nerve channels affecting the cortico-spinal tracts exclusively must cause the same symptoms, whatever its situation between the cortex cerebri and the pyramid decussation. In order, therefore, to diagnose with certainty a lesion of the brain-stem, still more to arrive at accurate conclusions as to the level at which it is situated therein, we have to depend upon the disturbances of function affecting the cranial nerves from the third to the twelfth, which in these cases are practically never absent.

As a matter of fact, an interruption of the cortico-spinal tracts alone, by a focal lesion within the brain-stem, is almost impossible. For these tracts, the central neurons for the motor spinal nerves have, mingled with them, as already stated, down to the point at which the medulla passes into the cervical cord, the central neurons for the motor cranial nerves. This condition is represented diagrammatically in Fig. 46, in which are given the cortico-bulbar tracts of the facial and hypoglossal nerves.

Let us consider the cases of two lesions, either of which causes an interruption in the pyramid tracts. If the interrupting lesion is situated in the medulla (A, Fig. 46), we shall have a lingual paralysis as well as that of the extremities, the facial muscles remaining unaffected. If, however, the lesion is in the pons (B, Fig. 46), the facial muscles as well as the tongue will be paralyzed. These cranial nerve paralyses, accompanying cortico-spinal paralyses of the extremities, proclaim, as do the latter, by the absence of degenerative atrophy and the reaction of degeneration, the supranuclear situation of the lesion which brings them about. We have, then, when endeavouring to form a diagnosis of the level of a lesion in the brain-stem, a very important principle of localization, in the fact that, in supranuclear lesions of the pyramids, the trophic influence of the cranial nerve nuclei remains intact, notwithstanding the paralysis of their corresponding muscles.

Foci, however, which affect a single tract only, as that of the pyramid, for instance, are, in a structure of small sectional area such as the medulla, of extreme rarity, and even in the more frontal portions of the brain-stem they are by no means common. We have, then, in the great majority of these cases, a mixed symptom complex, and here again symptoms of cranial nerve disturbance, accompanying those of interference with the long tracts, form the index to the localization of lesions of the brain-stem. We have seen how closely packed is the brain-stem with the nuclei of the cranial nerves. Small wonder, then, that in lesions of that structure one or other of the nuclei is almost always damaged. It is not necessary that the focus of disease shall actually include them within its area. Neighbouring foci may indirectly, by the action of

pressure, by causing circulatory disturbances and so on, bring about serious impairment of a nucleus. The paralysis, however, which follows the destruction of a motor nucleus in the brain-stem presents always the peripheral character—is accompanied, that is to say, by degenerative atrophy and the reaction of degeneration. So soon, therefore, as these conditions are found in the facial

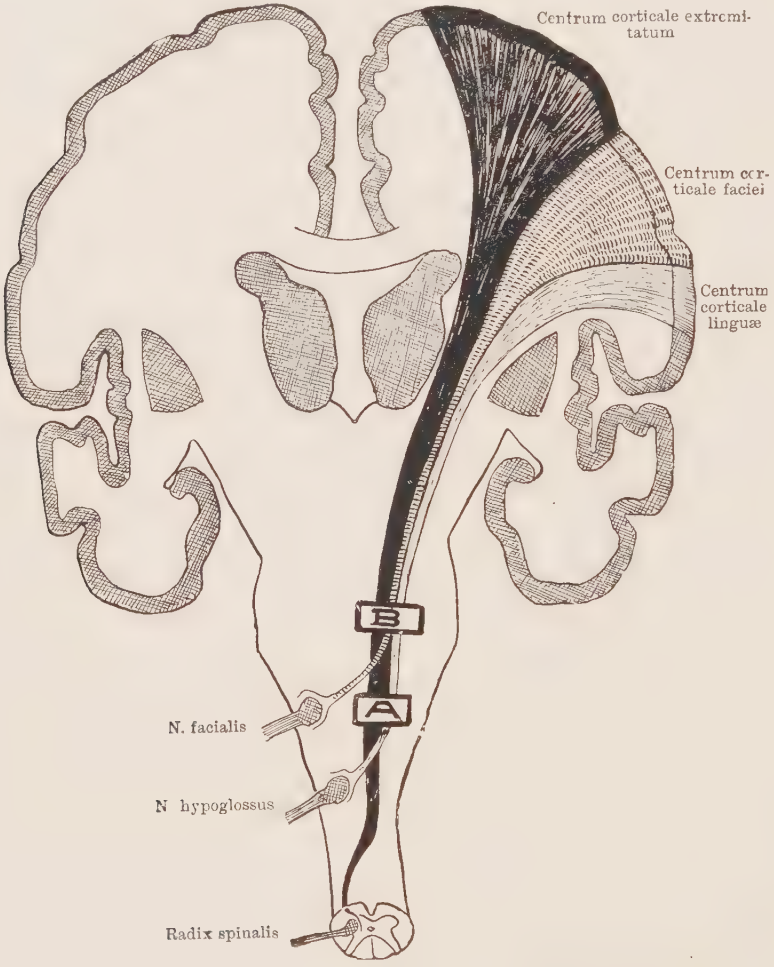


FIG. 46.—SUPRANUCLEAR PARALYSES OF CRANIAL NERVES IN PYRAMID LESIONS AT DIFFERENT LEVELS IN THE BRAIN-STEM.

muscles, the muscles of mastication or deglutition, the lingual or laryngeal muscles, a supranuclear situation for the lesion may be at once excluded. That in any given case the paralysis of particular nerves furnishes the most valuable diagnostic aid of all in establishing the level of the lesion goes without saying. A tegmental lesion which produces degenerative facial paralysis, but

spares tongue, œsophagus, larynx, and masseters, must be situated about the level of our section (Fig. 41)—*i.e.*, of the caudal extremity of the pons—and cannot extend either frontally to the region of the trigeminus or caudally to that of the nucleus ambiguus and the hypoglossal nucleus.

A further guide, therefore, in determining the level of a lesion of the brain-stem is afforded by the degenerative paralysis which affects those motor cranial nerves whose nuclei are involved in, or are in the very near neighbourhood of, the lesion.

Atrophic paralyses of muscles supplied by cranial nerves may, however, be brought about, not only by lesions affecting those nerves in their nuclei or roots, but also by lesions which involve them in their course to the periphery. A further criterion comes into play here—namely, the manner in which atrophic cranial nerve paralyses are combined with symptoms due to interruption of the long tracts in their course through the brain-stem. In these cases we frequently meet with some form of the condition known as *hemiplegia alterna*,* in which, as shown in Fig. 47, we have cranial nerves affected on one side, and a simultaneous interruption of motor and sensory tracts supplying more distally situated regions.

We may distinguish several types of hemiplegia alterna:

(*a*) In lesions of the cerebral peduncle we have, in addition to the pyramid lesion, which causes a complete hemiplegia of the opposite side (that is to say, one in which the facial muscles, the tongue, and the muscles of the extremities are affected), an involvement of the roots of the third nerve, which leads to an oculo-motor paralysis on the same side as the lesion (hemiplegia alterna oculo-motoria, or Weber's paralysis, Fig. 47, A).†

(*b*) In lesions of the caudal half of the pons we may have a *hemiplegia alterna facialis* produced in an analogous manner (Millard-Gubler's paralysis). In this condition we have facial paralysis on the same side as the lesion, and paralysis of the extremities on the opposite side (Fig. 47, B).‡ If the area of the lesion is large enough to involve the sixth nerve or abducens, as well as the facial of the same side, we have the condition known as "Foville's paralysis." In this condition we have also as a rule inability to look towards the side of the

* "Alterna" is to be preferred to the more frequently employed "alternans," which, as an active participle, is here out of place.

† Lesions which, without disturbing the pyramid tracts, exert an irritating action on the pars basilaris pontis by contiguity, and at the same time interrupt the fibres of the oculo-motorius, evoke motor irritative phenomena (tremor) in the opposite extremities, with homo-lateral oculo-motor paralysis. This is known as "Benedikt's symptom complex." As will be plain from Fig. 44, lesions affecting one of the red nuclei are specially likely to bring about the development of this syndrome.

‡ If the facial is not paralyzed, but merely irritated, we have homo-lateral facial spasm with contra-lateral paralysis of the extremities, a condition known as Brissaud's symptom complex. This syndrome, however, does not help towards a decision of the question whether the lesion is within or without the pons.

lesion; for an explanation *vide infra* in the section "Nerves supplying the Muscles of the Eye."

(*c*) A rare condition, whose infrequency is sufficiently explained by the smaller calibre of the brain-stem at the level of the medulla, is that of *hemiplegia*

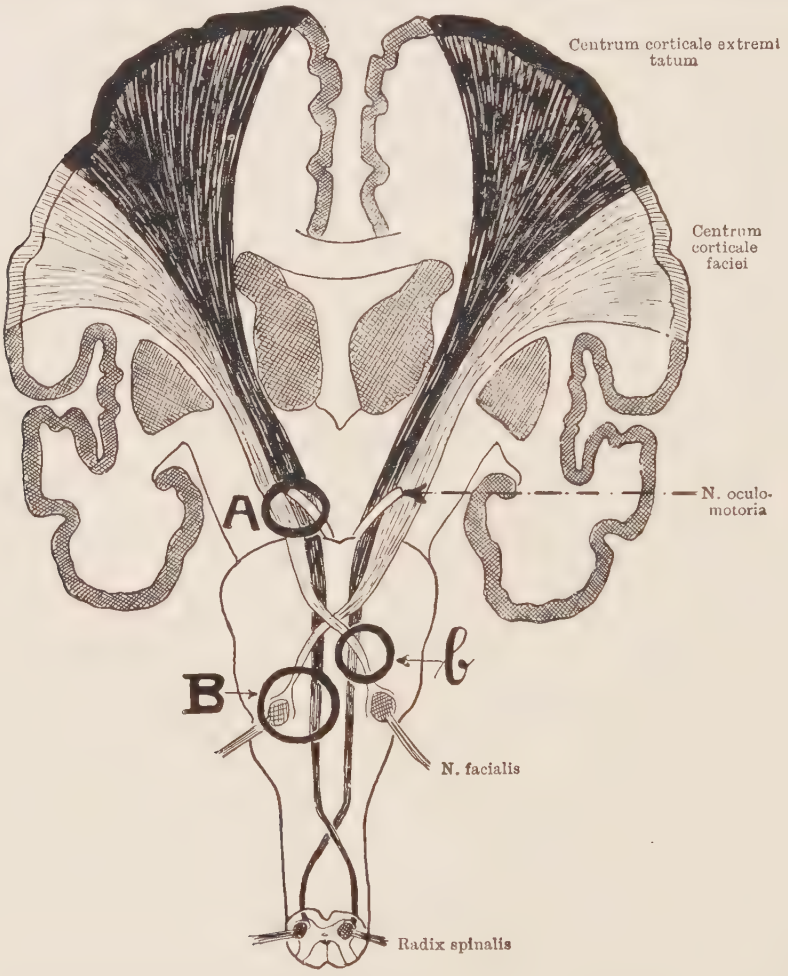


FIG. 47.—MODE OF ORIGIN OF ALTERNATING HEMIPLEGIAS.

A=lesion in the crus cerebri giving rise to hemiplegia alterna oculo-motoria; B and *b*=lesions in the pons giving rise to hemiplegia alterna facialis—in B with, in *b* without, degenerative atrophy of facial muscles.

alterna hypoglossica, in which half of the tongue is paralyzed on the same side as the lesion, and the extremities on the opposite side—so-called Jacksonian paralysis.

While it is the rule that, in these "alternating" symptom complexes, the cranial nerve paralysis is of the peripheral, degenerative type, cases do occur,

in the facial form (Millard-Gubler type), for instance, in which it, as well as the paralysis of the extremities, is supranuclear in type. The explanation of this fact can be gathered from a glance at *b* (Fig. 47), where the lesion is seen to be so situated that neither the facial nucleus nor its issuing root is involved, but, instead, the central facial tract shortly before its entry into the nucleus, and thus immediately after it has crossed the middle line and entered on the side of the lesion.

The trigeminus also, as well as the motor cranial nerves, may take part in an alternating symptom complex, in which loss of sensation over one side of the face, including the corresponding mucous membranes, is combined with hemiplegia of the opposite side. In such cases one will attempt to localize the lesion according to the motor cranial nerve paralyses which accompany the hemiplegia; for a facial anæsthesia of the kind may, so far as it alone is concerned, be produced by any lesion of the brain-stem caudal to the trigeminal nucleus. This possibility is due to the existence of the spinal or descending root of the fifth and the substantia gelatinosa. Lesions of these structures cause, it is true, no such extensive disturbances of sensation as are produced by those involving the chief sensory nucleus or the root fibres which enter it. It is probable, indeed, that destruction of the substantia gelatinosa in the medulla causes sensory disturbances only in the region supplied by the first branch of the trigeminus. We cannot make this statement categorically, however, as, at present, observations on the point are neither numerous nor conclusive enough to enable us to do so. Where masticatory paralysis is also present, the situation of the lesion may be placed, without hesitation, on the affected side, and not at a lower level than the middle third of the pons.

General corporeal sensibility may also be involved in one of these alternate symptom complexes. The lesion, for instance, to which a hemiplegia alterna is due may involve the fillet, in which case the hemiplegic portions of the trunk and extremities exhibit sensory disturbances in addition, or, again, the lesion, while sparing the pyramids, may involve, simultaneously, both the fillet and the cranial nerve nuclei or roots.

We have therefore to note, as a chief characteristic of lesions of the brain-stem, a generally "alternating," or rather "alternate," character of the motor disturbances present. Further, from the point of view of "transverse" or systemic diagnosis, the special peculiarities of the clinical picture presented by any given case of alternating hemiplegia may give us quite unambiguous information concerning, not only the side on which the lesion lies, but also its more ventral or more dorsal situation and its extent—this according to the number of cranial nerves involved, according to the involvement of pyramid or fillet, or of both, and so on.

Some account may be given here of the more important remaining deductions commonly used in the systemic diagnosis of affections of the brain-

stem. That affections of the tegmentum (pontis), and such other affections (*e.g.*, tumours of the vermis) as injure the tegmentum by pressure from without; that such affections manifest themselves chiefly by signs of injury to the cranial nerves goes without saying, for it is in the tegmentum that these have their nuclei. In these cases the dorsal longitudinal bundle is, owing to its dorsal situation, apt also to be involved. If this important association tract is only slightly involved, we have nystagmus developed on looking to the injured side; if the destruction is complete, combined movement of the eyes towards the side of the lesion becomes impossible (conjugate paralysis). We shall return to these facts and their explanation later on.

In so far as we have to do, in lesions of the tegmentum pontis, with injury to the motor nuclei, the nuclear character of the lesion is, of course, betrayed by the degenerative character of the paralysis which ensues. If the lesion extends to the deeper regions, sensory disturbances of the trunk and extremities are superadded (fillet). Finally, should it extend still farther in a ventral direction, the pyramids become involved (spastic paralysis of the musculature innervated from points distal to the lesion).

No less important is a scrupulously careful observation of the order in which the symptoms of nerve involvement develop, where there is in question the diagnosis of some morbid process at the base of the brain, such as a tumour growing into the brain-stem or an aneurysm of the basilar artery pressing upon it. In the latter case, the first symptoms likely to be observed (as will readily be deduced from anatomical considerations) are those of more or less symmetrical bilateral involvement of the pyramids, to which are added, later, signs of involvement of the fillet, and nuclear paralyses of cranial nerves other than the third and sixth (oculo-motor and abducens), which, owing to the mesial emergence of their roots, may be the first structures to present symptoms of disturbance. It is otherwise with new growths of the base of the brain, whose starting-point is generally the emerging nerve trunks or the meninges in their neighbourhood. Here, therefore, a unilateral symptom complex is the rule, and injury to nerve roots is the most significant, and almost always the earliest, symptom.

In these cases we often meet with "irritative symptoms," which exhibit a close analogy with those discussed when dealing with the spinal cord. They generally take the form of hyperæsthesiæ and "root pains" in the area of the trigeminus. The "irritative symptom" of the motor portion of the trigeminus is trismus, that of the facial nerve, spasmodic contractions of the facial muscles. We have also, fairly frequently, such sensory irritation phenomena as tinnitus, vertigo, etc. These points, as also the general symptomatology of the nerves of special sense of the brain-stem, which has been purposely left out of consideration up to the present, will be dealt with in the next section. In affections of the base of the brain (circumscribed meningeal inflammations must here

be considered, as well as tumours, gummata, and tuberculous deposits), the facial, abducens, and oculo-motor are the nerves most frequently affected. Of the last named, the fibres to the levator palpebræ are apparently the most vulnerable, for oculo-motor involvement generally makes itself known, in the first instance, by ptosis. Destruction of the pyramids is exceedingly rare; symptoms of slight impairment of their function come on gradually (spastic conditions, Babinski's reflex, etc.). Only in the region of the cerebral peduncle have lesions of the base any strong tendency to cause destruction of the pyramids, so that, in these lesions, *hemiplegia alterna oculo-motoria*, or *Weber's syndrome*, is often observed.

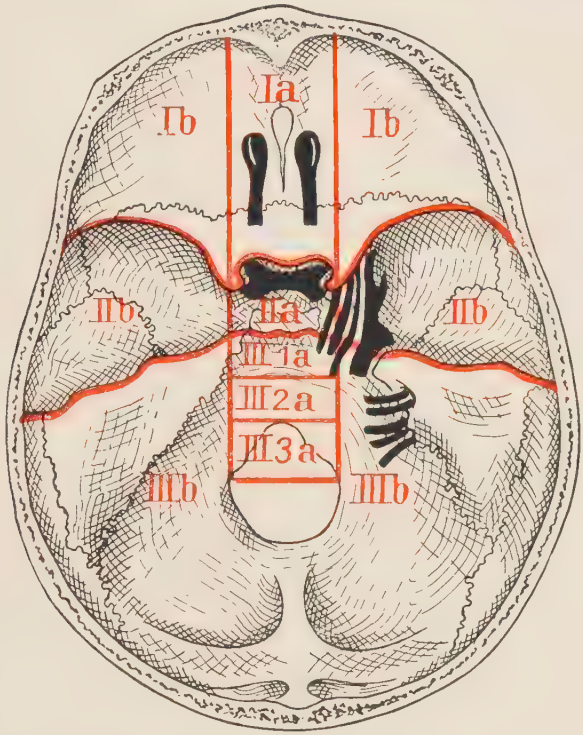


FIG. 48.

Topographical division of the base of the skull. The three cranial fossæ (I., II., III.) and their subdivisions; see Table on p. 100.

Though we have spoken of "irritative" affections of the nerves of the brain-stem as characteristic of basal lesions, we must not lose sight of the fact that they may, though much more rarely, accompany internal lesions of the brain-stem, such as hæmorrhages. Even the cerebellar irritative phenomena which we shall have to discuss later may be sometimes observed as distant effects of lesions in the pons and medulla.

In the following table we have endeavoured to represent in a topographical grouping the symptomatology of basal lesions. In making use of the table it

is advisable to refer to Figs. 33 and 48, and also to the illustration "Topography of the Cranial Base" (Fig. 100) in the section "Hypophysis."

Symptomatology of Lesions at the Base of the Brain.

	Median Lesion.		*Lateral Lesion.*	
Cranial Fossæ.	*Paralytic or Irritative Phenomena affecting Certain Cranial Nerves.*	*Other Symptoms and Typical Syndromes.*	*Paralytic or Irritative Phenomena affecting Certain Cranial Nerves.*	*Other Symptoms and Typical Syndromes.*
I. Anterior	Olfactory nerve	—	—	Moria (frontal lobe)
II. Middle	Optic nerve. *Trunk:* Amaurosis, skotoma. *Chiasma:* Heteronymous nasal or bitemporal hemianopia. *Tract:* Homonymous lateral hemianopia with hemianopic pupillary immobility without overlapping field of vision	Acromegaly, degeneratio adiposo-genitalis (hypophysis), hemiplegia, tetraplegia (peduncle)	Oculo-motor, trochlear, trigeminus, abducens	Henianosmia, parosmia (cortical olfactory centre)
III. Posterior	Oculo-motor	Weber's paralysis, Benedikt's syndrome (oculo-motor + peduncle)		
1. Peduncular portion	—	—		
2. Pontine portion	Trigeminus, abducens, facial, auditory	Hemiplegia, tetraplegia (pyramid tracts); Millard-Gubler's paralysis, Brissaud's syndrome (pyramid tracts + facial); Foville's paralysis (pyramid tracts + facial + abducens).	Facial, auditory, glossopharyngeal, vagus, accessory	Cerebellar ataxy, hypotonus, asynergia, adiadochokinesis etc. (cerebellum)
3. Bulbar portion	Facial, auditory, glossopharyngeal, vagus, accessory, hypoglossal	Hemiplegia, tetraplegia (pyramid tracts); Avelli's syndrome, Schmidt's syndrome (glosso-pharyngeal + vagus + (?) hypoglossal + (?) accessory); Jacksonian paralysis (pyramid tracts + hypoglossal); hemianæsthesia (fillet)		

Only a few words need here be devoted to the subject of lesions of the corpora quadrigemina. As we have here, crowded together in a small space, a number of important structures (oculo-motor and trochlear nuclei, median

and lateral fillets, the red nucleus of the tegmentum, which has important relations both with the cerebellum and the spinal cord, and finally, as we shall see later, an important portion of the optic tract), lesions in this region cause, simultaneously, pupillary paralysis, paralyses of the external muscles of the eye (frequently symmetrical and affecting separate corresponding muscles on the two sides), ataxia, deafness, disturbances of vision—many and varied symptoms, in fact, which we shall discuss in detail later on.

An early symptom of pressure on the region of the corpora quadrigemina (as a consequence, for example, of tumours in the anterior portion of the cerebellum, or in the posterior portion of the third ventricle) is vertical, upward nystagmus, of which we shall have more to say later. As a rare symptom may be mentioned the so-called "nystagmus retractorius," in which the eyeball is alternately retracted into the orbit and again advanced. The onset of symptoms of excitation of the sexual system, abnormal growth of hair and development of fat, abnormal height and premature development of the genital organs in young individuals, points to the pineal gland or epiphysis as the seat of a tumour pressing on the corpora quadrigemina. This syndrome is regarded as the expression of an alteration in the internal secretion of the pineal gland ("dyspinealism").

We cannot more appropriately conclude this section of our work than by the diagrammatic representation, in transverse sections of the brain-stem, of a few concrete cases of focal lesions, with their clinical symptoms appended. A study of the diagrams and of the accompanying table will serve as a brief and concise recapitulation of the foregoing material.

II. POINTS OF IMPORTANCE, FROM THE POINT OF VIEW OF LOCALIZATION, IN THE SYMPTOMATOLOGY OF THE NERVES OF THE BRAIN-STEM, WITH SPECIAL REFERENCE TO THE DIFFERENTIAL DIAGNOSIS OF PERIPHERAL AND CENTRAL LESIONS.

It will be necessary, seeing that signs of disturbance of the cranial nerves from the third to the twelfth are of such marked significance in the regional diagnosis of lesions of the brain-stem, to deal in still greater detail with certain points in their symptomatology. The discussion must include, in the first place, isolated features of importance and significance in the niceties of localization, which we have been obliged up to now to leave out of consideration, and, in the second, others which are of weight from the broader point of view of differential diagnosis. We must, in the case of a lesion affecting nerves of the brain-stem, but situated externally to that structure, avoid, so far as possible, the mistake of overlooking its peripheral character and regarding it as central. Finally, it will not be superfluous to describe, more exactly than we have been able to hitherto, the clinical phenomena corresponding to lesions of the cranial nerves.

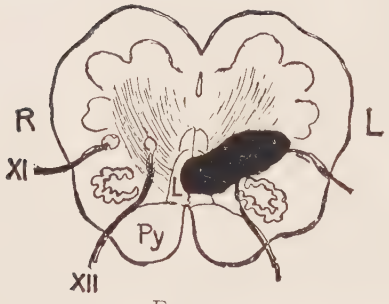


FIG. 49.

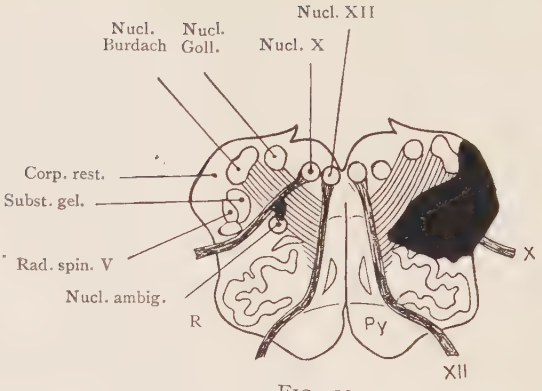


FIG. 50.

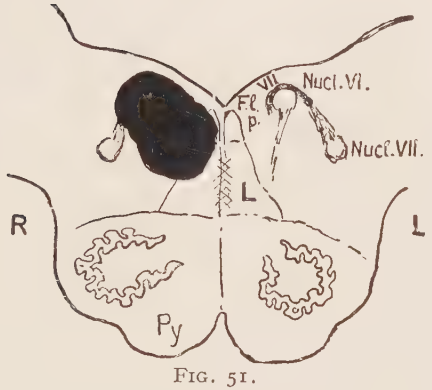


FIG. 51.

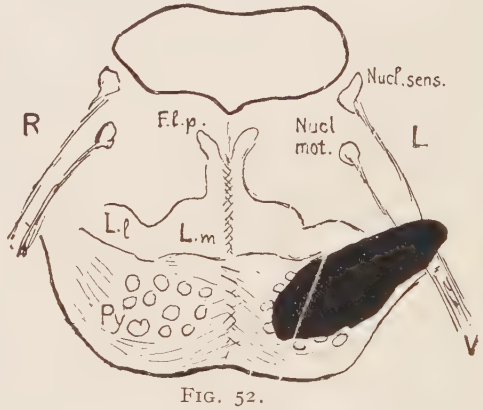


FIG. 52.

FIG 53.

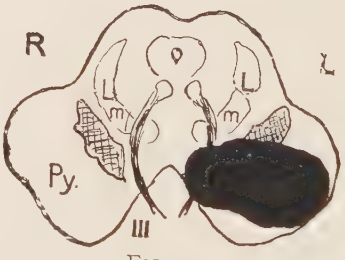


FIG. 54.

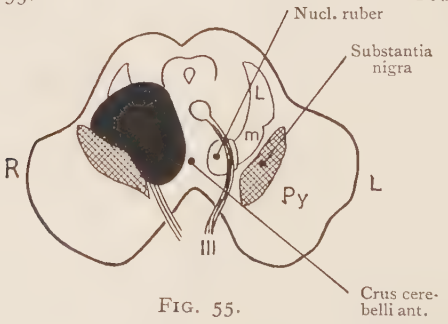


FIG. 55.

FIGS. 49-55; see Table on opposite page.

		Right.	*Left.*
Fig. 49	Head	—	Paralysis of accessory and hypoglossal nerves (degenerative)
	Trunk and Extremities	Ataxia and superficial anæsthesia	Ataxia
		Motility intact	
Fig. 50	Head	—	Anæsthesia in sphere of trigeminus. Palatal, pharyngeal and laryngeal paralysis (vagus)
	Trunk and Extremities	Analgesia and thermo-anæsthesia	—
Fig. 51	Head	Paralysis of facial and abducens nerves (degenerative); conjugate paralysis	—
	Trunk and Extremities	—	Disturbances of superficial sensibility.
		Motility intact	
Fig. 52	Head	Paralysis of facial and hypoglossal nerves (not degenerative)	Trigeminal anæsthesia and degenerative paralysis of the masticatory muscles
	Trunk and Extremities	Hemiplegia	—
		Sensibility intact	
Fig. 53	Head	—	Paralysis of facial and hypoglossal nerves (not degenerative)
	Trunk and Extremities	—	Hemiplegia. Sensory disturbances
Fig. 54	Head	Paralysis of facial and hypoglossal nerves (not degenerative)	Oculo-motor paralysis (degenerative)
	Trunk and Extremities	Hemiplegia	—
		Sensibility intact	
Fig. 55	Head	Oculo-motor paralysis	Trigeminal anæsthesia
	Trunk and Extremities	—	Hemianæsthesia (median fillet), choreo-athetotic movements (nucleus ruber)

In accordance with the foregoing, therefore, the nerves of the brain-stem may be grouped as follows:

(*a*) The caudal group: Glosso-pharyngeal, vagus, accessory, hypoglossal.
(*b*) Nerves of the cerebello-pontine angle: Auditory and facial.
(*c*) Trigeminus.
(*d*) Nerves of the ocular muscles: Oculo-motor, trochlear, abducens.

(*a*) The Caudal Group.

Let us first describe in their order the symptoms caused by lesions of nerves in this group.

The hypoglossal may be briefly described as the nerve supplying the muscles of the tongue. Its share in the innervation of the sterno-hyoid, sterno-thyroid, and omo-hyoid muscles is merely apparent. In the so-called "ansa hypoglossi," by which this innervation is effected, course no fibres from the brain-stem, but only fibres from the uppermost cervical nerves which have gained the hypoglossal trunk by anastomosis. Bilateral hypoglossal paralysis causes, of course, total glossoplegia: motionless, obeying gravity merely, the tongue lies sunken on the floor of the mouth; speech is unintelligible, and deglutition and mastication very difficult. In unilateral paralysis (hemiglosso-plegia), on the other hand, the disturbance of motility is relatively trifling; eating and speech are either not affected at all, or only slightly so. This is, of course, due to the marked reciprocal intertwining of the muscle fibres of the two halves of the tongue. If, however, the tongue is protruded, one notices, in consequence of the partially unopposed action of the genio-glossus of the sound side, a deflection of its tip towards the paralyzed side.

The accessory nerve supplies, unaided, the sterno-mastoid muscle, while, in its innervation of the trapezius, it is assisted by the upper cervical nerves. Destruction of the accessorius, therefore, causes complete paralysis of the sterno-mastoid. In unilateral lesions, then, we have inability to draw the chin fully over to the opposite side, while in bilateral lesions there is a tendency for the head to fall backwards. The paralysis of the trapezius is, on the contrary, not complete, and betrays itself chiefly by weakness and incompleteness in extension of the arm.

The vagus is a mixed nerve. On the one hand, it supplies the muscles of the palate, pharynx, larynx, trachea, bronchi, œsophagus, stomach, and small intestine with motor fibres, sends inhibitory fibres to the heart, and vaso-motor fibres to numerous bloodvessels; on the other, it is the sensory nerve for the dura mater, the external auditory meatus, the lower part of the pharynx, the larynx, the bronchi, the œsophagus, and the stomach. In these organs, therefore, terminate the fibres from the cells of the two vagal ganglia, the ganglion jugulare and the ganglion nodosum vagi, which are to be regarded

as analogues of the ganglia of the sensory spinal nerves. A complete bilateral paralysis of the vagus has, as will be obvious from the foregoing, no symptomatology, because it causes instant death. On the other hand, both partial bilateral and complete unilateral paralysis of the vagus may come under clinical observation. In the latter case we shall find unilateral paralysis of the palate, pharynx, and larynx. One-half of the soft palate hangs flaccid, with the result that the voice is nasal in character. One vocal cord is immobile in the median or cadaveric position, for the muscles which close, as well as those which open, the glottis are put out of action. Owing to powerful compensatory efforts on the part of the other vocal cord, the voice may remain normal; generally, however, it is somewhat hoarse, and is apt to break into a falsetto. The interference with deglutition is, on the other hand, almost always of a trifling character, for hemipharyngoplegia is, in consequence of the fact that the fibres of the pharyngeal muscles of one side interlace with those of the other, of no great functional significance. Tachycardia, or quickening of the heart's action, is but rarely observed in unilateral vagus lesions, and the same may be said of respiratory disturbances, such as slowing and irregularity of respiration. In incomplete vagal paralyses only some of the symptoms above mentioned are present, and in some cases these only partially developed. Thus, instead of complete paralysis of a vocal cord, we may have merely a paralysis of the cricoarytenoideus posticus, whose function is to open the glottis. The result of this "posticus paralysis," especially when bilateral, is interference with respiration, phonation remaining unaffected. The converse condition, aphonia with normal respiration, seems only to flourish on the soil of hysteria.

The glosso-pharyngeal is an almost exclusively sensory nerve, whose cells are situated in the ganglion superius and ganglion petrosum glosso-pharyngei. According to the latest researches, only a small motor portion is, in addition, to be credited to it, which portion supplies the stylo-pharyngeus, an elevator of the pharynx. The glosso-pharyngeal conveys sensory impressions from the upper part of the pharynx and from the middle ear, as also gustatory perceptions from the palate and the posterior third of the tongue. Loss of taste in the latter regions is accordingly the most important symptom of its interruption; anæsthesia of the pharynx may be detected, but especially noticeable is the abolition of pharyngeal and palatine reflex, which, however, is only of significance for localization if unilateral. Bilateral abolition of these reflexes occurs, as is well known, in purely functional affections. The stylo-pharyngeus is of so little importance as an elevator of the pharynx, a function which it shares with the pharyngo-palatine muscle, that no motor disturbance of importance is to be expected as the result of a paralysis affecting it alone.

As regards irritative symptoms in the region of the caudal group of nerves, they are one and all without real importance from the point of view of localization. Some of them are to be referred, in almost every case in which they

occur, to the neuroses, and do not justify the diagnosis of an organic irritative lesion of the affected structures. This is especially true of the different forms of glossospasm, tonic and clonic, and of spasms of the muscles supplied by the accessorius (spasmus nutans, etc.). Pharyngismus, and laryngismus and œsophagismus also, are hardly ever to be referred to a localized lesion of the vagus. When these conditions do not depend on a general functional or organic nervous disease (hysteria, tetany, tabes, hydrophobia, tetanus), they are as a rule reflex phenomena due to disease of the affected portions of the respiratory and digestive tracts. On the other hand, we are perfectly justified in regarding bradycardia, Cheyne-Stokes respiration, and cerebral vomiting as irritative symptoms originating in the vagus centres in the medulla. (For example, the vomiting caused by injections of apomorphine fails to take place if, as in bulbar paralysis, etc., the vagus nucleus has itself suffered damage, and, conversely, the bradycardia due to vagus irritation—in contrast to that present in lesions of the atrio-ventricular bundle in the heart—may be controlled by an injection of atropine.) Nevertheless, significance from the point of view of localization must be denied also to these morbid phenomena; they may, in consequence of the apparently extraordinary sensitiveness of the vagus nucleus, come on as the result of any intracranial affection which causes pressure on the brain, whatever be its actual situation. They are therefore generally—and rightly—reckoned among the symptoms, not of focal, but of general diseases of the brain.

If, then, the irritative symptoms are to be withdrawn from our consideration, it becomes all the more important, where symptoms pointing to morbid processes in the region of the cranial nerves IX. to XII. are present, to determine where interruption of their continuity has taken place—whether between the cerebrum and the medulla (supranuclear,), or in the region of the bulbar nuclei (nuclear), or, again, beyond these, in the peripheral portion of the nerve's course (infranuclear). In the latter case it is practicable, and, of course, immensely important, to distinguish between intracranial and extracranial lesions.

Supranuclear lesions are, so far as motor symptoms are concerned, to be diagnosed first and foremost from the absence of degenerative atrophy and the reaction of degeneration in the affected muscles. Importance is also to be attached to the determination of an increased irritability to mechanical and electrical stimuli. These points are of general application. A special imprint is given to supranuclear paralyses of the nerves we are now considering by the fact that their cortico-bulbar innervation is a bilateral one. Each cerebral hemisphere, that is to say, shares in the innervation of the nuclei of both sides. This is particularly the case with the vagus, and, in consequence, unilateral interruptions of the tracts leading from the cortex to the nucleus ambiguus present no clinically demonstrable disturbances, inasmuch as the one hemi-

sphere remaining in unbroken communication with the nuclei of both sides suffices to maintain in functional efficiency the pharyngeal and laryngeal muscles on the side of the lesion as well as on the opposite side (*vide* Fig. 56).

If, then, we have before us a unilateral pharyngo- and laryngoplegia we may, without waiting for an electrical test, conclude against the supranuclear nature of the paralysis. It is almost the same with the accessorius and the hypoglossus. Thus it will be found that, in cerebral hemiplegias, the functions of the sterno-mastoid are generally as little affected as are those of the glottis

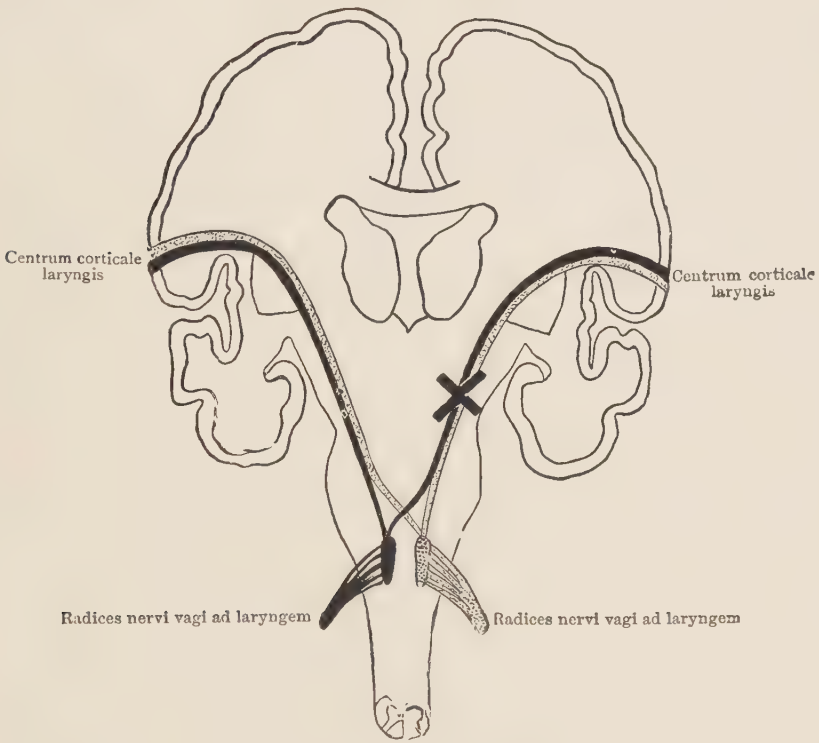

Centrum corticale laryngis
Centrum corticale laryngis
Radices nervi vagi ad laryngem
Radices nervi vagi ad laryngem

FIG. 56.—THE LARYNGEAL CENTRE IN THE NUCLEUS AMBIGUUS.

Diagram showing bilateral supranuclear innervation.
(A unilateral supranuclear lesion X cannot cause laryngeal paralysis.)

and the mechanism of deglutition. Here then, also, we have complete bilateral innervation from the cortex. The trapezius, it is true, usually only escapes unilateral paralysis so far as its upper or clavicular portion is concerned. Only this portion, which is distinguished as the respiratory portion, seems, therefore, to share in the bilateral cortical innervation, the remainder of the muscle being dependent not only on the accessory, but also on the upper cervical nerves. Finally, unilateral nuclear and infranuclear paralysis of the hypoglossal causes no serious disturbance of motility, a consequence, as stated on p. 104, of the

reciprocal intertwining of muscle fibres of the two halves of the tongue. In the case of supranuclear lesions, the fact that each hypoglossal nucleus is innervated from both cerebral hemispheres (though predominantly from the opposite cortex) tells in the same direction. It is therefore not to be wondered at that, in cases of the latter kind—cerebral hemiplegia, for example—the functional disturbances in the region of the hypoglossal are very trifling, and, as a rule, only reveal themselves in a deflection of the protruded tongue toward the paralyzed side.

Bilateral interruption of their supranuclear tracts causes, on the contrary, as will be plain from the foregoing, a bilateral paralysis or paresis of the nerves under review, with serious motor impairment in the pharynx, larynx, and tongue. This impairment resembles that observed in lesions of the bulbar nuclei. The symptoms have therefore been spoken of as "pseudo-bulbar paralytic." They may be brought about in two ways: (1) By simultaneous symmetrical interruption of cortico-bulbar tracts. (2) Unilateral interruption, which, owing to the bilateral innervation of the affected nuclei, leaves the functions of the latter undisturbed, is complicated later on by a similar interruption on the other side, and immediately "pseudo-bulbar" symptoms manifest themselves, of course, on both sides. Cases of pseudo-bulbar paralysis with only unilateral lesions have been described, but they are of extreme rarity.

It will help to the presentation of a clear and coherent account of the symptom complex known as "pseudo-bulbar paralysis" if we allow ourselves here a slight digression. We will anticipate somewhat by stating that the motor trigeminus, thus the masticatory musculature, has, like the vagus, a bilateral cortical innervation; further, that unilateral interruption of the cortico-bulbar tract for the lowest portion of the facial (orbicularis oris) causes a hemiparesis of the latter, but only a bilateral interruption can produce a disturbance of the function of the lips, as organs of speech, at all approaching in severity that observed in bulbar paralysis (*vide infra*). The nervous structures just mentioned, then, suffer in pseudo-bulbar paralysis an impairment of function quite analogous to that observed in the caudal group of oblongata nerves. The clinical picture presented by pseudo-bulbar paralysis (also called "glosso-pharyngo-labial paralysis") is, in consequence, extremely characteristic, and is constituted as follows: Speech is drawling or dragging, monotonous, sometimes aphonic. Consonants are pronounced badly and with difficulty, so that in severe cases speech is unintelligible. Vowel formation is less seriously interfered with. The breath is often lost while speaking, so that several attempts have to be made before a sentence is completed, and speech is semi-explosive. According as the paralysis of the lips or that of the palate is predominant, so we have prevailing either difficulty in forming labials or a nasal quality of voice. The motility of the tongue may be so impaired

that it lies motionless on the floor of the mouth; generally, however, a certain power of protrusion is left, but the movements to one side or the other or the power of curving the sides inwards to form a tube are affected, or repeated protrusion soon leads to inability to continue the movement. (In many cases the interruption of the bilateral cortical tracts is only partial.) The movements of the palate are either abolished or are slow and incomplete; in the larynx, paresis of the vocal cords seldom reaches the more severe degrees. The muscles of mastication are generally more markedly affected; it is impossible to clench the teeth firmly, and the mouth often remains half open. The paralysis of the pterygoids reveals itself by incapacity for protrusion and lateral movement of the lower jaw; occasionally the muscles which open the mouth are also paralyzed, and it is impossible to open the mouth completely or against resistance. Very significant symptoms are often noticed when the attempt is made to take food. Here, in addition to the weakness of the masticatory muscles, that of the muscles of the tongue, lips, and cheeks plays an important rôle. The food cannot be pushed between the rows of teeth, falls out of the mouth, has to be passed to the back of the mouth with the help of the fingers, and so on. If, in addition, there is paralysis of the palate and pharynx, portions of food find their way into the larynx and the nose. In the less severe cases, however, especially if the patient eats slowly and chooses solid or only semi-liquid foods, the act of eating is carried out fairly well. The essential difference between these conditions and those found in true bulbar paralysis is that in the former the paralyzed muscles exhibit neither degenerative atrophy nor the reaction of degeneration. It is, in fact, *mutatis mutandis*, the same difference as exists between spinal progressive muscular atrophy and spastic spinal paralysis. A condition of genuine contracture may, as a matter of fact, gradually supervene in the lips, tongue, or palate of patients suffering from pseudo-bulbar paralysis. Exaggeration of the masseter reflex is hardly ever absent, and requires no explanation here.

We pass now to the nuclear paralyses of the caudal group of nerves, which, as we have seen, are readily distinguished from the supranuclear. The reaction of degeneration is easily demonstrated in the tongue, the sterno-mastoid, and separate bundles of fibres of the trapezius; with somewhat more difficulty in the palate and pharynx. In the larynx only a highly skilled specialist will succeed in demonstrating it with certainty; in practice we must content ourselves with the demonstration that powerful electrical stimulation of the recurrent laryngeal nerve in the neck causes no adduction of the vocal cords. In the tongue, which of all muscles is the most easily inspected and palpated, we soon notice loss of volume, abnormal wrinkling of the surface, softness, flabbiness, and fibrillary twitchings.

Not so simple is it to distinguish nuclear from infranuclear lesions—that is to say, from those affecting the nerves under consideration at some point or

other of their peripheral course. Electro-diagnostic tests for the reaction of degeneration and the search for degenerative atrophy alike fail us here utterly, for, so far as those phenomena are concerned, it is immaterial whether the nucleus is affected or its corresponding nerve trunk at some part of its peripheral course. If the nature and situation of the injury or disease do not betray at once the infranuclear character of the paralysis present, we must depend on a consideration of the remaining symptoms for guidance. The medulla is so small in volume that an isolated nuclear paralysis of either the glosso-pharyngeal, vagus, accessory, or hypoglossal nerve is hardly ever seen. Where, however, we have uncombined affections of these nerves, motor and sensory disturbances, for the most part of the type of "alternating" paralyses, are generally also present. Further, the two hypoglossal nuclei (right and left) are so close to one another that a nuclear lesion generally causes bilateral paralysis of the tongue. A fact of great importance is that, in nuclear as distinguished from infranuclear lesions of this nerve, the hypoglossal paralysis is combined with a paresis of the orbicularis oris of the same side. We must conclude, therefore, that in the innervation of the orbicularis oris, not only the facial, but also the hypoglossal nucleus participates, the latter sending off fibres within the medulla to join the facial trunk.

Combined paralyses of the various nerves of the caudal group may result also from lesions whose situation is infranuclear, but intracranial (tumours, aneurysms, exudates, etc., in the posterior cranial fossa). We find the characteristic symptom complex of palatal, laryngeal, and lingual paralysis; the sterno-mastoid and trapezius are sometimes affected,* but the orbicularis oris remains intact. For the rest, the basal situation of the lesion is indicated by the order in which the symptoms develop (*vide* p. 98).

After their exit from the cranial cavity, the conditions necessary for the simultaneous destruction of several of the nerves under review are very rarely found. The morbid process must have its seat high up in the neck, and will generally lend itself readily to direct inspection, palpation and Roentgenoscopy. As regards lesions affecting single members of the caudal group of nerves at some point of their peripheral course, these generally cause only partial paralyses, as the nerve branches proximal to the seat of the lesion of course escape. Thus, disturbances of the mechanism of deglutition are very rarely found in extracranial affections of the vagus; if the nerve is injured in the mediastinum (by tumours, aortic aneurysm, etc.), the picture presented is generally one of paralysis of the recurrent laryngeal only—immobility of the vocal cord in the mid-position. Exactly the reverse is true of the hypoglossal nerve. If it is affected at or below the point at which the fibres from the upper

* A pharyngo-laryngeal or glosso-pharyngo-laryngeal hemiplegia is known as Avelli's "syndrome"; if to it is added a homolateral paralysis of the sterno-mastoid and trapezius, the symptom complex is known as Schmidt's.

cervical roots join the ansa hypoglossi, we have symptoms which are otherwise foreign to the picture of hypoglossal paralysis—namely, paralyses of the external laryngeal muscles which are innervated from the ansa (sterno-hyoid, sterno-thyroid, and omo-hyoid), recognizable by the degenerative atrophy of the muscular layer covering the thyroid cartilage, and by the lateral displacement of the larynx in the act of swallowing.

(*b*) The Nerves of the Cerebello-Pontine Angle.

(α) *The Facial Nerve.*

This nerve supplies all the facial muscles—including the buccinator, but excepting the levator palpebræ superioris, which is supplied by the oculomotor nerve—from the occipito-frontalis down to the platysma myoides, and, in addition, the stylo-hyoid, the posterior belly of the digastric, and the stapedius.

A lesion interfering with the motor function of the facial nerve causes facial paralysis, which, when unilateral, is known as "monoplegia facialis," when bilateral as "diplegia facialis." It is also known as "Bell's paralysis." The paralyzed side of the face is masklike, immobile, expressionless; the naso-labial fold is smoothed out; the forehead cannot be wrinkled; the eye, in consequence of the paralysis of the orbicularis oculi, cannot be closed (*lagophthalmos*); the angle of the mouth hangs downwards. The falling of the base of the tongue betrays the paralysis of the stylo-hyoid and posterior belly of the digastric, an abnormal acuity of hearing, and special sensitiveness to deep tones (*oxyakoia, hyperakusis*), that of the stapedius, whose function it is to close the fenestra ovalis of the tympanic cavity with the plate of the stapes.

The facial trunk, however, brings with it from its place of origin other centrifugal fibres—namely:

1. *Lachrymal Fibres.*—These fibres leave the facial trunk as it passes through the cranial wall (Fallopian canal), coming off from the geniculate ganglion as the *nervus petrosus superficialis major*, and joining the *nervus lachrymalis trigemini*.

2. *Salivary Fibres.*—These form a complex known as the "*nervus intermedius* of Wrisberg" or as "Sapolini's nerve" (*cf.* also *infra*, p. 125). These fibres also leave the facial trunk in the Fallopian canal, but at a point only just above the stylo-mastoid foramen. They also join the trigeminal system, the resulting anastomosis forming the *chorda tympani*, through which the salivary fibres of the facial reach the *nervus lingualis trigemini*.

Between the points at which these two nerves (*petrosus superficialis major* and *chorda tympani*) leave the facial trunk, a further bundle of nerve fibres joins it, which belongs strictly to another nerve, the trigeminal, and only runs for a short distance with the facial, returning then to the trigeminus. Its fibres are centripetal; they are, in fact, the gustatory fibres from the anterior two-thirds

of the tongue. From the lingual they pass along the chorda tympani to the facial trunk in the Fallopian canal, where they join the motor and salivary fibres of the facial to form a common trunk. They pass upwards to the geniculate ganglion, which they enter. The latter does not therefore, strictly speaking, belong to the facial, for centrifugal nerves have no ganglia.* From

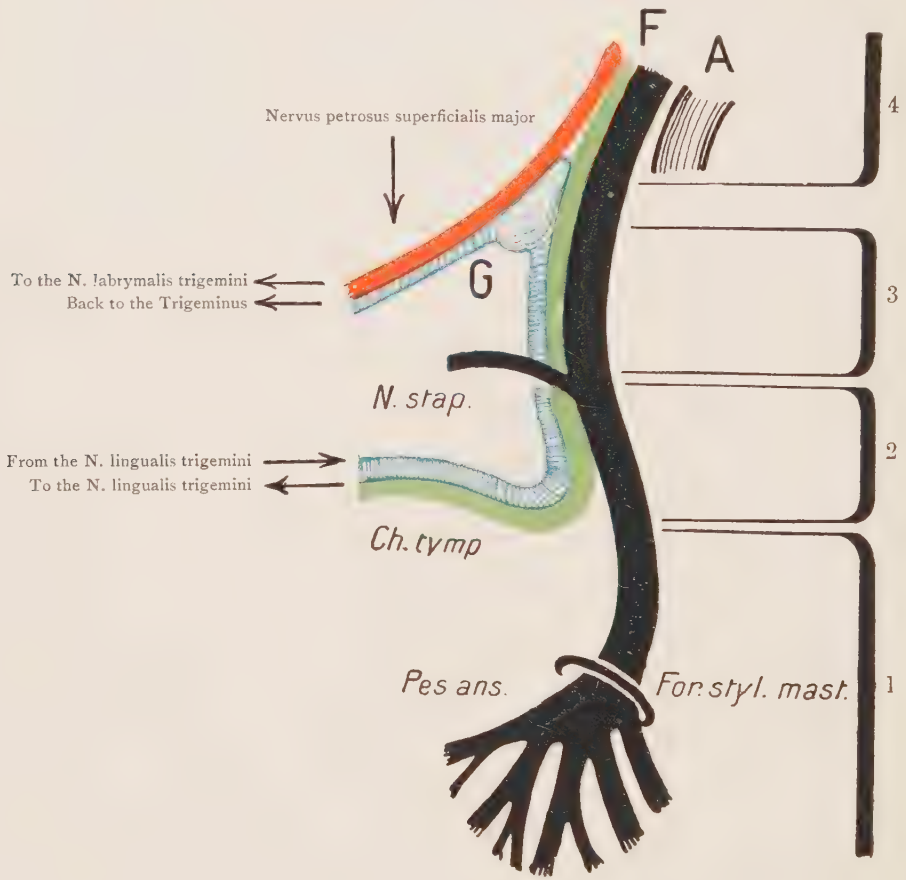


FIG. 57.—SCHEMA SHOWING THE COURSE OF THE VARIOUS GROUPS OF FIBRES IN THE FACIAL TRUNK

A.=Acusticus. F.=Facialis=motor fibres, lachrymal fibres, salivary fibres.
G.=Gangl. genic=gustatory fibres.

the ganglion these gustatory fibres, accompanied by the lachrymal fibres of the facial, pass along the superficial petrosal nerve to the trigeminus again. If they are involved in a lesion during their short passage along the facial trunk, the clinical sign of their interruption (loss or diminution of taste in the anterior

* According to the view which classes the *N. intermedius* as a mixed nerve, containing gustatory as well as secretory fibres (*vide* note, p. 125), the geniculate ganglion must be regarded as belonging partly to the intermedius.

two-thirds of the tongue) accompanies, though foreign to the function of the facial, the ordinary signs of facial paralysis—"caught together, hanged together."*

These peculiarities of the peripheral course of the facial and its several components are represented diagrammatically in Fig. 57. The central, supranuclear course of the nerve also presents some rather complicated features:

1. Those portions of the facial nucleus from which the upper facial fibres arise—*i.e.*, the frontal and palpebral branches—are innervated, like the laryngeal centre in the nucleus ambiguus represented in Fig. 56, from the cortex of both cerebral hemispheres. The frontal muscles present a true type of bilateral synergetic action; the orbicularis oculi also is, under ordinary circumstances, always contracted simultaneously on both sides; the separate closing of one eye is, as is well known, an action that has to be specially learnt. The lower facial, on the other hand (mouth and cheeks), has a crossed cortical innervation; in eating, mimicry, etc., corresponding muscles on the two sides are very often quite unsymmetrically employed.

2. The hypoglossal nucleus sends some fibres to the portion of the facial which supplies the oribicularis oris. When, therefore, a labial paresis is associated with nuclear paralysis of the hypoglossal, it is not necessary to assume an affection of the facial, unless other signs of the same are present.†

Fig. 58 illustrates the supranuclear innervation of the facial.

The anatomico-physiological conditions presented by the facial as above described (*a*) in its central, (*b*) in its peripheral course enable us to diagnose, from the clinical picture presented in any given case, the point at which an interruption of continuity in the facial system has taken place. We have endeavoured to represent the matter in tabular form. A recollection of what has just been stated, and an occasional reference to Figs. 57 and 58, will make the table intelligible, and render superfluous any detailed explanation thereof.

The irritative phenomena observed in the region of the facial—spasmodic contractions, twitchings—have, as a rule, no significance from the point of view of localization; for in the great majority of cases they are of functional, neurotic character (tetany, spasmodic tics, etc.), or are reflex phenomena having their originating stimulus in painful affections of the face, mouth, etc. Even if we assume an organic cause for the spasmodic contractions in the region of the facial which are observed in the initial stage of progressive paralysis, we have

* In regard to vaso-motor fibres in the facial trunk and others connected with the sweat function, *vide infra* in the section on the Trigeminus, where, also, the question of the entry of gustatory fibres from the chorda tympani into the nuclear region of the *N. intermedius* is discussed (note, p. 125).

† Connection between the hypoglossal nucleus and the orbicularis oris may also be re-established by peripheral anastomoses. In a case of total destruction of the facial trunk through caries of the petrous bone I observed a partial restitution of function in the orbicularis oris which could only be explained in this way.

as yet no certain knowledge concerning the material lesion on which they may depend. On the other hand, spasmodic facial contractions have been observed as the result of pressure on the intracranial facial trunk by tumours, aneurysms, etc. (*e.g.*, as a part of Brissaud's symptom complex). They may also be pro-

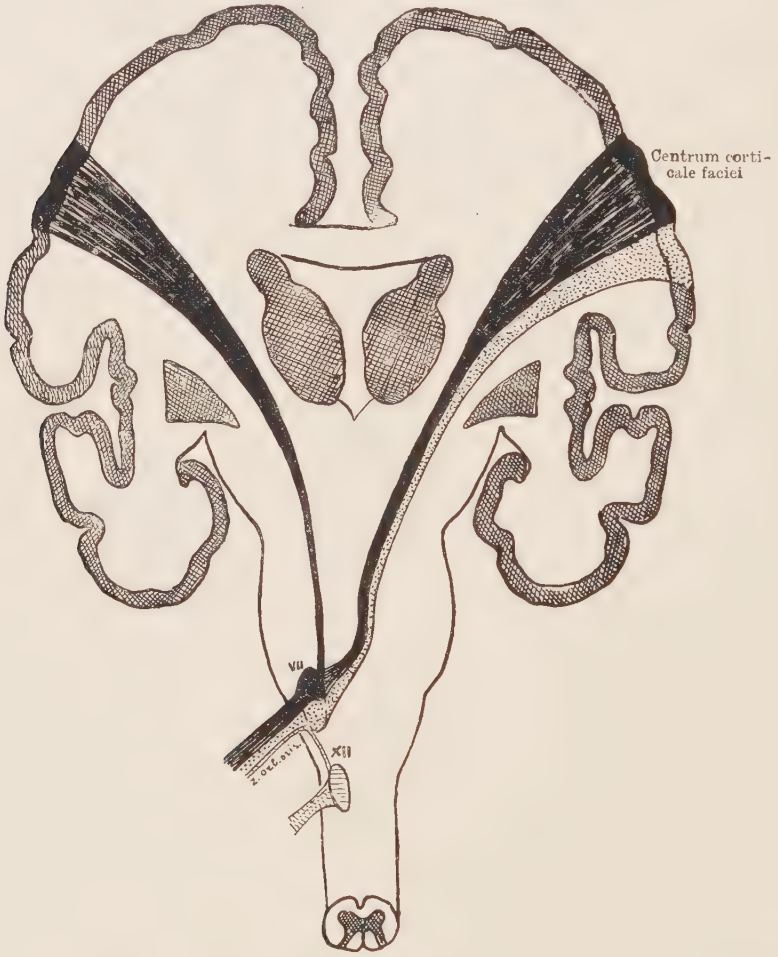


FIG. 58.—SCHEMA SHOWING CENTRAL INNERVATION OF THE FACIAL NERVE.

= Upper facial.

= Lower facial.

duced by irritation of the nerve in its peripheral course, due to contracting scars on the face—*e.g.*, duelling wounds (*Mensurverletzungen*). Only exceptionally is heightened mechanical irritability of the facial, as exhibited in the so-called Chvostek's phenomenon, to be observed under these conditions. As

a rule this symptom (twitchings in the region of the facial on tapping or even on stroking the skin over the pes anserinus) has a functional basis, as, for instance, in tetany. Finally, we shall see later, when we come to discuss the subject of cortical epilepsy, that such irritative symptoms are a frequent consequence of irritative lesions of the facial centre in the cortex. In late stages also of incomplete facial paralyses, of whatever causation, spasmodic contractions may come on in the paralyzed muscles.

REGIONAL DIAGNOSIS OF LESIONS OF THE FACIAL NERVE.

A. Lesions of the Peripheral Portion of the Facial.

GENERAL CHARACTERS:

1. Generally unilateral.
2. Diminution of electrical excitability; reaction of degeneration.
3. The whole facial musculature affected as a rule.

SPECIAL CHARACTERS:

I. *Infranuclear Lesions.*

(*a*) *External to the Cranial Cavity.*

Monoplegia facialis is the affection present in almost every case (exception: bilaterial otitis).

(*α*) *Distal to the Branching off of the Chorda Tympani* (Fig. 57, 1).—Symptoms: Prosopoplegia. If the lesion is quite peripheral—*i.e.*, beyond the pes anserinus—some of the facial branches may escape.

(*β*) *In the Fallopian Canal between the Chorda and the Branch to the Stapedius* (Fig. 57, 2).—Symptoms: Prosopoplegia; loss of taste in anterior two-thirds of tongue; diminution of salivary secretion.

(*γ*) *In the Fallopian Canal between the Branch to the Stapedius and the Geniculate Ganglion* (Fig. 57, 3).—Symptoms: Prosopoplegia; loss of taste in anterior two-thirds of tongue; secretion of saliva diminished; hyperakusis.

(*δ*) *Between the Internal Auditory Meatus and the Geniculate Ganglion* (Fig. 57, 4).—Symptoms: Prosopoplegia; no loss of taste; salivary secretion diminished; frequently nerve deafness through participation of auditory nerve; where this is not the case, hyperakusis; suppression of lachrymal secretion, emotional and reflex.

(*b*) *Within the Cranial Cavity—Basal Lesions.*

Diplegia facialis frequent (basal gummatous meningitis).

Symptoms: As (*δ*) above; generally coexisting affections of a number of basal nerve roots (abducens, glosso-pharyngeal, vagus, accessory, hypoglossal); general cerebral symptoms (vertigo, vomiting, headache).

II. *Nuclear Lesions.*

Distinguishing Features: Abducens almost always simultaneously affected, as also pyramids (Millard-Gubler's paralysis, *vide* p. 96 and Fig. 47, B) or fillet (hemianæsthesia alternans, *vide* p. 97); in a large proportion of cases the upper facial escapes.

B. Lesions of the Central Portion of the Facial.

GENERAL CHARACTERS:

1. Almost without exception unilateral.
2. No diminution of electrical excitability.
3. Upper facial unaffected or only very slightly involved (bilateral innervation, *vide* pp. 113-115, Fig. 58).

SPECIAL CHARACTERS:

I. *Lesions in the Cerebrum, Cerebral Peduncle, and Frontal Portion of the Pons.*

Distinguishing Features: Combination with paralysis of the extremities on the same side (in consequence of the proximity to each other of the central facial neurons and the pyramid tract).

II. *Lesions in the Caudal Portion of the Pons, Prenuclear Lesions.*

Distinguishing Feature: Combination with crossed paralysis of the extremities (*vide* p. 96 *et seq.*, and Fig. 47, B).

(*b*) *The Auditory Nerve.*

In the section on the Structure of the Brain-Stem different portions of the cochlear and vestibular mechanism have been dealt with separately as they have come under our notice. We must now briefly consider, in its continuity, the course followed by each of these components of the auditory nerve.

The cochlear nerve originates from cells in the spiral ganglion of the cochlea. These are bipolar cells whose peripheral extensions are in connection with the auditory cells of the organ of Corti, while their central extensions enter the cochlear nucleus in the cerebello-pontine angle. Here, with connecting neurons of the second order, begins the central auditory tract. The

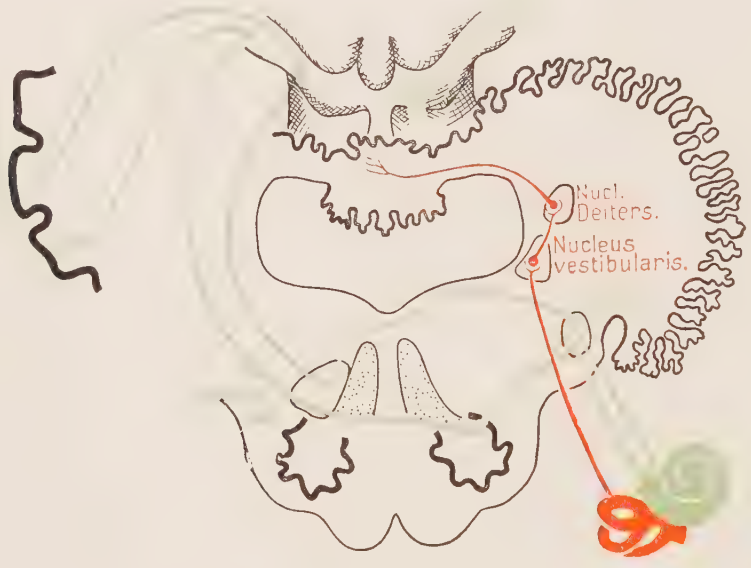


FIG. 59.—DIAGRAM OF THE AUDITORY TRACTS.
Cochlear fibres, green; vestibular fibres, red.

manner in which this passes across to the fillet of the opposite side is shown in Fig. 59. The fibres from the dorsal portion of the cochlear nucleus (acoustic tubercle) make their way under the floor of the fourth ventricle, where they form the *striæ medullares* or *acusticæ;* those from the ventral portion pass through the base of the tegmentum pontis, where, in the olive, connection is made with fresh neurons. These latter fibres are known as the "*fibres of the trapezium.*" Both sets of fibres, after reaching the fillet, pass in the form of the lateral fillet and fillet of Reil to the posterior corpus quadrigeminum and its intimately related corpus geniculatum mediale, from which structures the terminal fibres of the whole neuron chain of the acoustic tract pass to the cortical auditory centre in the temporal convolutions.

The vestibular nerve has its origin in the cells of the vestibular ganglion, which are connected peripherally with the sensory epithelium of the ampullæ, utricle, and saccule. Its terminal ramification takes place in the vestibular nucleus. The cells of the latter, however, send fibres to the neighbouring nucleus of Deiters of the same side, from which they finally pass to the vermis cerebelli (Fig. 59).

As is plain from the foregoing, the vestibular mechanism is in the closest anatomical connection with the cerebellum. Their co-operation, also, in the maintenance of equilibrium and the sense of position in space, establishes between them so close a physiological unity that the symptomatology of the vestibular system must be left to be dealt with later, in the section on the Cerebellum.

Here, therefore, we will concern ourselves only with the actual nerve of hearing, the cochlear nerve. Impairment of the function of the cochlear brings with it, of course, deafness, partial or complete. As, however, deafness may be due to affections of the conducting apparatus of the middle and outer ear, which are entirely within the province of the aurist, our first task will be to consider carefully the characters distinctive of the so-called "nerve" deafness.*

The two most important distinguishing features of nerve deafness are loss or diminution of the ability to hear by conduction through the bones of the skull, and diminution in the power of tone perception in the higher ranges. On the other hand, deafness due to abnormalities of the conducting apparatus (membrana tympani, ossicles) is distinguished, as is well known, (1) by a lengthened and strengthened perception of sounds conducted through the bones of the skull, and (2) by a diminution in the power of tone perception in the lower ranges.

The acuteness of perception by means of bony conduction is most easily investigated by determining the length of time during which (*a*) the patient, (*b*) the normal-hearing observer can perceive the sound of a vibrating tuning-fork, the handle of which is placed against the vertex, the teeth, or the mastoid process. This is known as *Schwabach's experiment.* If nerve deafness is present, we have a shortening of the period during which the sound can be perceived by bony conduction, or, if the deafness be complete, a total abolition of such perception. In affections of the middle ear, on the other hand, the tuning-fork, applied after Schwabach's method, is heard for a longer period than normal. This test can, of course, only be applied diagnostically if the affection of hearing is bilateral.

Other tests easily and quickly applied are Rinne's and Weber's tests.

* Between nerve deafness (*sensu strictiori*)—namely, that due to lesions of the cochlear nerve or its supranuclear tracts—and that due to lesions of the percipient structures in the cochlea, no symptomatological distinction can be drawn. From the neurological point of view, therefore, the regional diagnosis of lesions of the auditory mechanism must be regarded as wanting in precision.

In Rinne's test a vibrating tuning-fork is held in contact with the mastoid process of the subject under examination; when he ceases to hear it there, it is held close to his ear. If the hearing is normal, the subject now hears the tuning-fork again. This is spoken of as the "*positive*" result of Rinne's test. In disease of the sound collecting and conducting apparatus of the ear, nothing is heard in this second stage of Rinne's test. This is the "*negative*" result. In nerve deafness, on the other hand, one generally obtains "*positive Rinne,*" unless of course the impairment of hearing has reached a high degree, in which case hearing by air conduction is also markedly impaired or abolished. Really reliable results, however, are given by Rinne's test only in cases of bilateral deafness; in unilateral nerve deafness the vibrations may be carried to the sound ear, and perceived there for a longer time than those conveyed by air conduction to the affected ear (so-called "*false negative Rinne*").

In Weber's test, the inferences connected with which are more clearly defined, a vibrating tuning-fork is applied to the vertex. A normal subject will hear the sound in both ears; if one ear be now stopped, he will "lateralize" the sound to the side on which air conduction has been thus broken. A patient with disease of the conducting apparatus "lateralizes" spontaneously to the affected side ("*positive Weber*"); while, on the other hand, the patient with nerve deafness will lateralize to the sound side ("*negative Weber*"). In bilateral affections Weber's test is of no assistance, nor does it help in cases of combined nerve deafness and middle-ear disease. One must also be on one's guard against misleading answers from such patients, who, instead of observing themselves objectively, are apt, by auto-suggestion, to "lateralize" to the sound ear, since they are convinced that "it must be so." The double possibility should therefore be made clear to them in advance.

Affections of tone perception are investigated by means of the so-called "Galton's whistle," or by "Struycken's monochord." Deafness for the higher notes is characteristic of affections of the labyrinth and auditory nerve tracts, as distinguished from those of the middle and outer ear.* In nervous deafness the hearing for words with sharp consonants and clear short vowel sounds is specially affected—*e.g.*, "sister," "sweats," "fish," etc.—while those with broader consonant and vowel sounds ("brood," "hundred," "oars," "dawn," "worm," etc.) are much better heard and understood. The opposite is the case in non-nervous deafness.

A really complete deafness is always a "nerve" deafness. In pure middle-ear disease there is always a residue of hearing for the lower tones.

That the diagnosis of nerve deafness is also made by exclusion, after examination of the membrana tympani, after finding that inflation by Politzer's

* In testing the hearing for higher tones it must, however, be borne in mind that these (in contrast to the lower), in cases where the hardness of hearing or deafness is unilateral, are perceived in the sound ear even when it is stopped; it is therefore necessary to cut it out by a noise-producing apparatus.

bag produces no improvement, etc., is of course well known. Opinions are still divided as to the value of the curious symptom known as "*parakusis Willisii*" in the diagnosis of nerve deafness. Many otologists hold that patients suffering from nerve deafness hear better in noisy places; others, however, maintain that the symptom may be present in uncomplicated middle-ear diseases.

The irritative symptoms to be referred to the cochlear nerve are of the nature of subjective noises in the ear (tinnitus aurium). The noise may be of a roaring, buzzing, or whistling character, and may be located by the patient in the interior of the ear, or may seem to be external. These subjective phenomena are of little help in localization, as they are present also in many cases of middle-ear disease. In ankylosis of the stapes, for example, there is generally severe tinnitus, due to a continuous irritation of the neighbouring organ of Corti. At the same time singing in the ears (the note is generally of very high pitch) is so constant an accompaniment of nervous deafness, that some authorities have held that, in any case in which there is no history of tinnitus, the deafness may be regarded, from that fact alone, as in all probability not nervous in character.

Regional diagnostic importance is attached by aurists to certain qualitative peculiarities which may characterize the subjective noises present in any given case. If due to middle-ear affection, they will vary hardly at all in intensity and tone level, whereas a marked variability in both of these qualities is characteristic of nerve deafness. Where, however, it is established that we have to deal with a case of nerve deafness, it is only possible to infer with certainty an affection of the auditory nerve trunk when accompanying symptoms point to a basal morbid process. In such cases a simultaneous affection of the vestibular nerve is most probable. (As will appear more fully later on, affections of the vestibular nerve reveal themselves by phenomena of the nature of vertigo.) The facial and other of the cranial nerves are likely, however, to be involved as well, and symptoms referable to the oblongata, the pons, and even the cerebellum, are likely to show themselves in a manner which gives a special character to affections of the cerebello-pontine angle, and somewhat facilitates their diagnosis.

We have here chiefly to deal with tumours, especially fibromata and fibrosarcomata, arising from the nerve sheaths of the facialis and acusticus at their emergence. The clinical picture developed by such tumours is distinguished by the following symptoms, for whose explanation later sections must be referred to (trigeminus, nerves of the ocular muscles, cerebellum, etc.): Inability to direct the eyes towards the side on which the lesion is situated, nystagmus, the movements being directed towards the affected side. Absence or diminution of reflexes and of sensation in the sphere of the fifth nerve, facial paresis, nervous deafness, adiadochokinesia on the affected side; choked disc, cerebellar,

ataxia and pains at the back of the head, often confined to the affected side, or more strongly developed there. Nystagmus and absence of corneal reflex

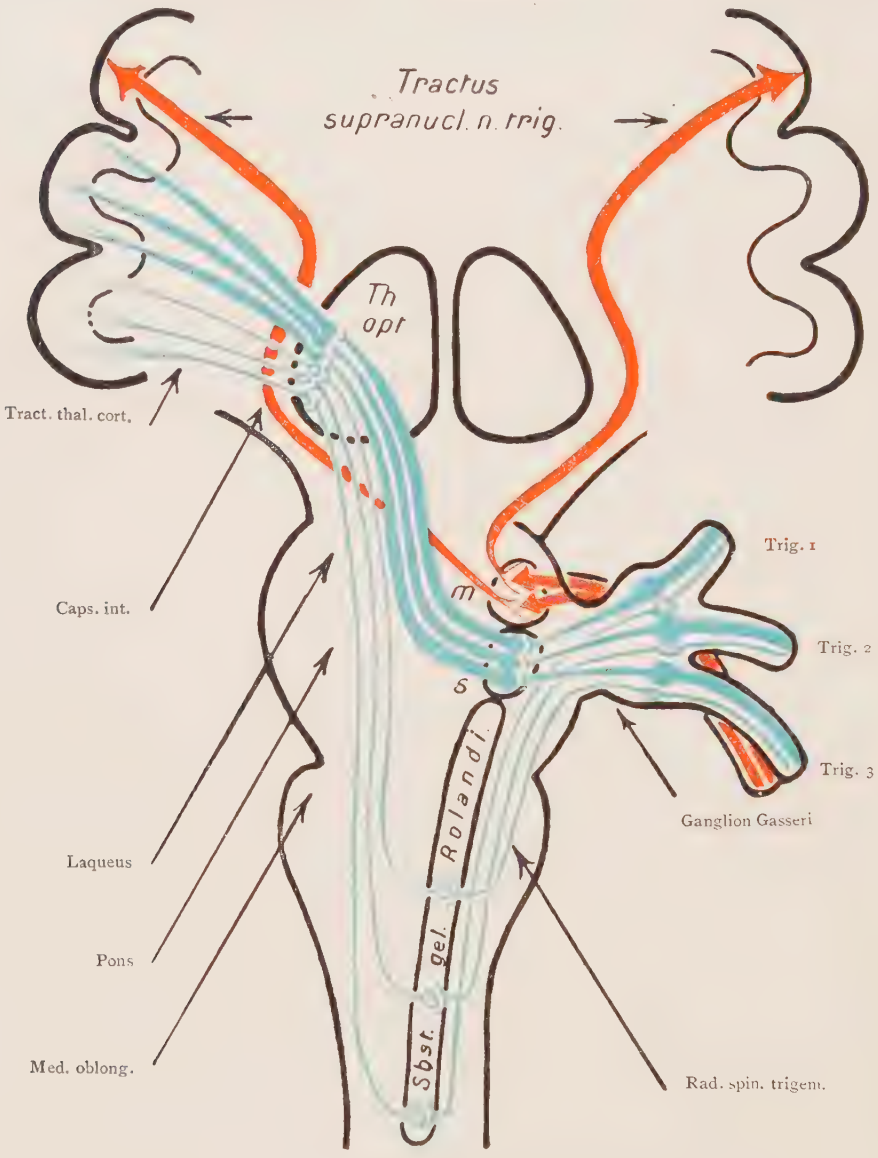


FIG. 60.—DIAGRAM OF THE CENTRAL TRIGEMINUS TRACTS.
Motor tracts, red; sensory tracts, blue. (Light=system of the substantia gelatinosa; broad=system of the sensory nucleus of the locus cœruleus.)

are in some cases not in evidence until the patient is made to lie on the side opposite to that of the tumour, so that the latter presses on the cerebello-pontine angle.

(*c*) The Trigeminus.

We know that the trigeminus has an anterior, motor, and a posterior sensory root. While the latter, in strict analogy with the posterior roots of the spinal cord, forms a ganglion—the ganglion of Gasseri—the anterior or motor root passes behind the ganglion to join the third of the three main branches which issue from it, and to confer on it, in so doing, the character of a mixed nerve.

In the Gasserian ganglion are situated the cells of origin of the fibres of the sensory root. Of these, part terminate, as is seen in Fig. 60, in the nucleus of the locus cœruleus, while part make their way downwards as the spinal root of the trigeminus, giving off as they go terminals which enter the nuclear column known as the *substantia gelatinosa*.

From the two sensory nuclei (*locus cœruleus* and *substantia gelatinosa*) the trigeminal neuron tracts of the second order pass across the middle line to the fillet of the opposite side, in which they pass to the thalamus. Here finally arises a tertiary neuron system which terminates in the cortical sensory centre for the trigeminus.

It can be seen clearly from Fig. 60 that the supranuclear or cortico-pontine innervation of the motor trigeminus is a bilateral one.

The structures innervated with sensory fibres from the three main branches of the trigeminus are:

(*a*) *The first or ophthalmic branch.*

(α) The cutaneous area, marked 1 in Fig. 61.

(β) Conjunctiva, cornea, and iris.

(γ) The mucous membrane of the frontal sinuses and the upper portion of the nose.

(*b*) *The second or maxillary branch.*

(α) The cutaneous area (2, Fig. 61).

(β) The mucous membrane of the antrum of Highmore and the lower portion of the nose.

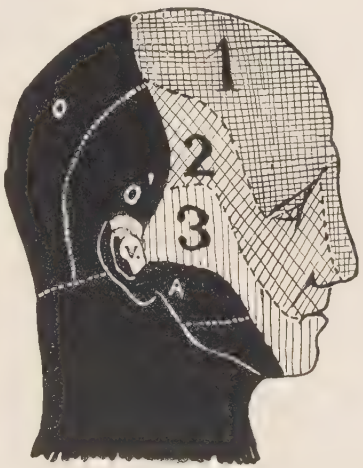


Fig. 61.—The Sensory Nerve-Supply of the Head.

Shaded: Trigeminus { 1. Ramus ophthalmicus. 2. Ramus maxillaris. 3. Ramus mandibularis.
White: Vagus. V. = Nervus auricularis vagi.
Black: Cervical nerves { O = N. occipitalis major. O′ = N. occipitalis minor. A = N. auricularis magnus.

(γ) The mucous membrane of the upper jaw, and the soft palate up to the pharyngeal arch.

(δ) The upper teeth.

(*c*) *The third or mandibular branch.*

(α) The cutaneous area (3, Fig. 61).

(β) The mucous membrane of the cheeks, lower jaw, floor of the mouth, and tongue.

(γ) The lower teeth.

Total or partial destruction of one of the three branches leads, of course, to anæsthesia or hypæsthesia in the structures innervated from that branch. Further, in lesions of the ophthalmic branch, the conjunctival and corneal reflexes are abolished, as also the sternutatory reflex evoked by irritating the upper part of the nasal mucosa; in lesions of the maxillary branch the sneezing reflex evoked by irritation of the lower nasal mucous membrane is abolished, as well as the anterior palatal reflex. It must be borne in mind, however, that the latter is inconstant, and is often absent in functional neuroses. The same is true of the conjunctival and corneal reflexes, and even the reflex excitability of the nasal mucosa is subject to individual variations. For this reason it is only to unilateral absence of these phenomena that decisive significance can be allowed. It should be remembered also that the pricking sensations felt on smelling ammonia or acetic acid are due to irritation of the sensory nerve endings of the trigeminus in the nasal mucosa, and have nothing to do with the olfactory nerve. They are absent in trigeminal lesions, as are also the reflex symptoms evoked by them (lachrymation, alterations in the pulse-rate, catching of the breath, etc.).

The trigeminus, in addition to its ordinary sensory functions, acts as a nerve of special sense. The mandibular, or rather one of its branches, the lingual nerve, conveys gustatory sensations from the anterior two-thirds of the tongue. We have described above (pp. 111 and 112) how its fibres pass, with the chorda tympani, to join the facial trunk, in which they pursue a short course, leaving it again and rejoining the trigeminal system. This reunion takes place in the spheno-palatine ganglion of the maxillary branch, with which branch the lingualis fibres pass into the Gasserian ganglion and the nuclear region of the trigeminus. The question whether they remain there, or whether they reach finally the glosso-pharyngeal, is of no clinical importance.

The structures innervated from the motor portion of the third branch of the trigeminus are:

(*a*) The muscles of mastication.

(*b*) The anterior belly of the digastric and the mylo-hyoid muscle.

(*c*) The tensor tympani and tensor veli palatini muscles.

Unilateral paralysis of the muscles of mastication is known as "monoplegia masticatoria." In this condition lateral movement of the lower jaw is only possible towards the paralyzed side, because only the pterygoids on the sound side can act. With a finger placed on the masseter and the temporal muscle, it is easy to satisfy oneself of the absence of contraction in those muscles. In diplegia masticatoria the lower jaw falls and all lateral movement ceases. Further, the mandibular reflex is abolished—the contraction of the masseter which can be evoked in most normal individuals by percussing the finger placed along the lower row of teeth.

Paralysis of the anterior belly of the digastric and of the mylo-hyoid will produce, in some cases, a flaccid condition of the floor of the mouth. Little is known of any special symptoms due to paralysis of the tensor tympani and tensor veli palatini. They remain latent in most cases. Occasionally, however, it would seem that we have to attribute to such paralysis anomalies in the position of the palato-pharyngeal arch, and dysakusis for deeper tones.

Irritative phenomena in the sensory sphere are—Pains, hyperæsthesiæ or anæsthesia dolorosa, the latter condition being analogous to that bearing the same name described in connection with the spinal cord (p. 30). In the motor sphere we have tonic and clonic spasms of the masticatory muscles. As regards the localizing significance of irritative symptoms in the domain of the motor trigeminus, broadly speaking, what was said in connection with the irritative symptoms of the facial on pp. 113 and 115 applies here also, save that here these phenomena are due less often to functional neuroses than to infective morbid processes (tetanus, meningitis, etc.). Pains in the region of the trigeminus, too, lose much of their significance from the frequency of genuine spontaneous neuralgias. At the same time the absence of neuralgiform phenomena in central affections of the fifth nerve deserves special mention.

Besides the motor and sensory fibres above discussed, all three branches of the trigeminus are joined, soon after their exit from the cranial cavity, by sympathetic fibres, which accompany the branches and some of their subbranches in their further course. The junction of these sympathetic fibres, arising from the plexuses accompanying the arteries to the head with trigeminus fibres, takes place at certain ganglia in the course of the latter. These ganglia are:

1. For the ophthalmic branch, the *ganglion ciliare;* its roots arise from the *plexus caroticus internus.*

2. For the maxillary branch, the *ganglion spheno-palatinum,* whose roots have the same origin.

3. For the mandibular branch, the *ganglion oticum,* whose roots spring from the *plexus meningeus medius.*

4. In addition, the chief offshoot of the mandibular, the lingualis, possesses a ganglion of its own, the *ganglion submaxillare,* whose sympathetic roots are drawn from the *plexus maxillaris externus.*

What, then, are the functions of these sympathetic fibres ?

In order to arrive at an answer to this question, we would refer in the first place to Fig. 31, and to the account given in that part of our work of the sympathetic symptom complex for the head (*vide* p. 60 *et seq.*). The sympathetic tracts depicted on the right of the figure—"ad musculum tarsalem superiorem," "ad musculum dilatatorem pupillæ," "ad musculum orbitalem" —pass, the first two through the ganglion ciliare, the last through the ganglion spheno-palatinum. A destructive lesion here may lead, in the one case to pupillary paralysis and narrowing of the palpebral aperture, and in the other to enophthalmos. The tracts "ad glandulas sudoriparas" and "ad vasa sanguinea" must, however, communicate with all three branches of the trigeminus. For in interrupting lesions of these nerves we find, in almost every case, in the anæsthetic parts, either heat and redness (early cases) or coldness of the surface and cyanosis (older cases), also anidrosis. It may be well to point out here that, occasionally, the same phenomena are noticed in cases of facial paralysis, so that it would appear that some of these sympathetic fibres may accompany the facial trunk.

Among other fibres accompanying branches of the trigeminus in their course, but of sympathetic origin, which may be mentioned, are the fibres governing the secretion of nasal mucus, which form part of the fibre complexes of the ophthalmic and maxillary branches. It is on interruption of their continuity that the abnormal dryness of the nasal mucosa depends, which accompanies paralysis of those nerves, and leads, secondarily, to impairment of the sense of smell.

We have already seen that the salivary fibres of the mandibular (lingual branch), and the lachrymal fibres of the ophthalmic (lachrymal branch), come from the facial trunk, the former *via* the chorda tympani, the latter *via* the greater superficial petrosal nerve. The connection between the lachrymal fibres and the first or ophthalmic branch of the trigeminus takes place through the above-mentioned spheno-palatine ganglion and an anastomosis between the first and second rami of the trigeminus, the zygomatic nerve. It follows from the foregoing that trigeminus lesions situated on the cerebral side of the zygomatic nerve—lesions of the Gasserian ganglion, for example—do not cause a drying-up of the lachrymal secretion. Lachrymal paralysis only occurs when the lesion affects the spheno-palatine ganglion itself, the immediately adjoining portion of the maxillary ramus, the zygomatic, or the lachrymal nerve —in a word, some part of the tract marked black in Fig. 62.

On the other hand, a trigeminus lesion which reveals itself by impairment of the salivary secretion can only be situated quite peripherally in the system of the third ramus, for it is through the junction of chorda fibres from the facial with the lingual nerve that the latter first acquires salivary functions.

Disturbances of taste in the anterior two-thirds of the tongue may, however, be found—

1. In lesions proximal to the spheno-palatine ganglion, provided that they involve the second ramus, the Gasserian ganglion, or the common trigeminal trunk.*

2. In lesions of the lingual nerve distal to the junction with the chorda tympani.

Where, however, such gustatory disturbances are found associated with diminution in the secretion of saliva, but without anæsthesia in the domain of the trigeminus or paralysis in that of the facial (*vide supra*, p. 115), the chorda tympani itself must be the seat of the lesion, a not uncommon eventuality in affections of the tympanic cavity.

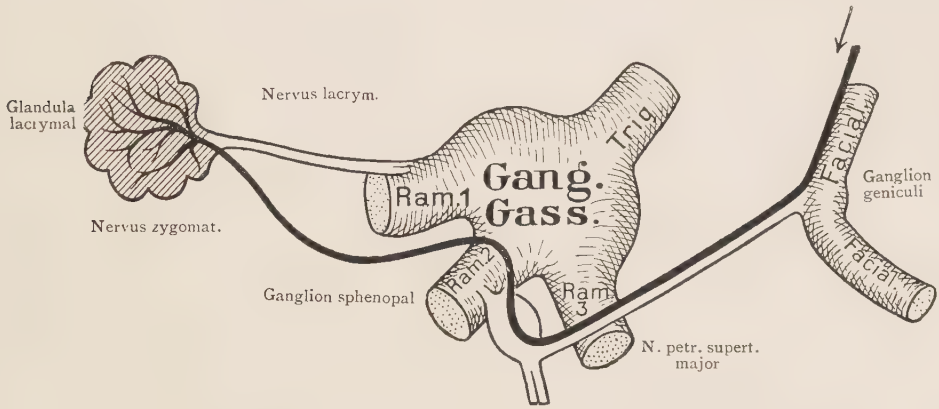


FIG. 62.—THE LACHRYMAL TRACT IN THE FACIALIS AND TRIGEMINUS.

It may be stated broadly that lesions of separate rami or of peripheral branches of the fifth pair are quite rare; apart from injuries to the bones or the soft parts of the face, we have as causes only tumours and tuberculous or syphilitic affections of the bones through which the nerves make their exit, or of their periosteal covering. The *ramus ophthalmicus* leaves the cranial cavity through the superior orbital fissure; the *ramus maxillaris* passes first through the foramen rotundum, later (as the infra-orbital nerve) through the canal of the same name; the foramen ovale gives passage to the *ramus mandibularis*, whose terminal branch, the inferior alveolar nerve, lies in the mandibular canal. The points of exit of the three branches on to the face are the supra-orbital, infra-orbital, and mental foramina respectively.

* As, after extirpation of the Gasserian ganglion, ageusia of the anterior two-thirds of the tongue is sometimes entirely absent, while in other cases the power of taste is merely diminished, the conclusion is justified that in such individuals a certain proportion of gustatory fibres from the geniculate ganglion, instead of passing to the trigeminus by way of the superficial petrosal nerve, join the intermediate nerve of Wrisberg (*vide* pp. 111 and 112), and enter its nuclear area. This portion of the facial must in these cases be regarded as a mixed (sensory secretory) nerve.

Much more frequently the Gasserian ganglion and the common trunk of the nerve are affected, almost always unilaterally (*meningitis gummosa*, aneurysms of the internal carotid, transverse basal fractures posterior to the sella turcica, tumours of the hypophysis, etc.). In such cases one finds, in addition to anæsthesia over the whole of one trigeminal region, *monoplegia masticatoria*, the peripheral origin of which is demonstrated by the onset of degenerative atrophy and the reaction of degeneration. The atrophic condition of the muscles shows itself in a falling in of the temporal fossa and the masseteric region, and, consequently, by an abnormal prominence of the zygomatic arch. The occurrence of herpes zoster in the cutaneous region of the fifth nerve points to affection of the Gasserian ganglion (*cf.* the analogous phenomenon in affections of the spinal ganglia; *vide* pp. 31 and 54).

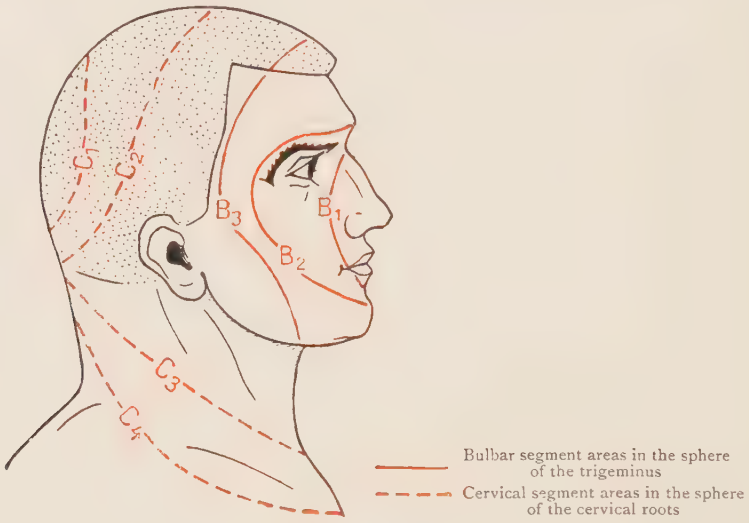


Fig. 63.—Spinal and Bulbar Segment Areas for the Sensory Supply of the Head.

At the base of the brain the trigeminal trunk and its ganglion are seldom affected alone by pathological processes, but almost always in company with other cranial nerves. Here also we have the same general distinguishing features as in other basal affections.

Nuclear trigeminal affections are generally associated, as has been stated, in a typical manner, with evidences of involvement of other structures in the brain-stem. Where lesions in the oblongata impair the functions of the trigeminus by involving the *substantia gelatinosa* or the spinal root of the nerve, the participation of other nerve roots and nuclei in the medulla in the alternating symptom complex points to the caudal situation of the trigeminus lesion.

It must here be noted that, in affections of the most distal portion of the

substantia gelatinosa in the medulla, we have disturbances of sensation in the frontal region; if the lesion be somewhat higher up, the temples and eyelids are affected; while a still more proximally situated lesion affects the nose and cheeks. Thus, nuclear trigeminal anæsthesias present localizing conditions distinguishing them from those of a peripheral nature, just as is the case with the spinal sensory nerves. Fig. 63, comparison of which with Fig. 61 is instructive, gives an approximate representation of these conditions, and shows the "onion layer" arrangement of the areas of sensory disturbance in the region of the trigeminus in lesions of the cervical cord. B_1, B_2, and B_3 are segment areas corresponding respectively to the upper, middle, and lower portion of the bulbar nuclear column of the trigeminus (*substantia gelatinosa Rolandi*). At the same time, in partial affections of the fifth nerve, even when the typical "onion layer" topography is absent, disagreement between the actual distribution of the symptoms and the innervation areas given in Fig. 61 must be taken as proof of the central situation of the lesion.

As regards supranuclear lesions of the trigeminus, they cause, if unilateral, no motor, but only sensory symptoms, because of the bilateral cortical innervation of the masticatory nuclei. The clinical picture of supranuclear masticatory diplegia has been already drawn in the discussion of pseudo-bulbar paralysis (pp. 108 and 109).

Thanks to the foregoing various points on which it is possible to make definite statements, the localization of morbid processes and of injuries in the course of the trigeminus and its branches presents, in actual practice, no great difficulties.

(*d*) The Nerves supplying the Muscles of the Eye.

We stated, in our preliminary remarks on the anatomy of the brain-stem, that there were three nuclei for the ocular muscles—those, namely, of the abducens, trochlear, and oculo-motor nerves.

While we have hitherto spoken of the latter as a single structure, we have now to consider the somewhat complicated conditions arising from the fact that the separate muscles supplied by the oculo-motor nerve are represented by separate nuclear groups in the area of origin.

The nuclear region of the oculo-motor contains, as is seen in diagram in Fig. 64—

1. A small-celled lateral nucleus, the nucleus of Edinger and Westphal.
2. A large-celled lateral nucleus.
3. A small-celled mesial nucleus.

Of these, the longest is the large-celled lateral nucleus. It contains, ranged one behind the other (the nuclei are represented in our figure in horizontal projection):

(1) The centre for the levator palpebræ superioris.
(2) ,, ,, rectus superior.
(3) ,, ,, rectus internus.
(4) ,, ,, obliquus inferior.
(5) ,, ,, rectus inferior.

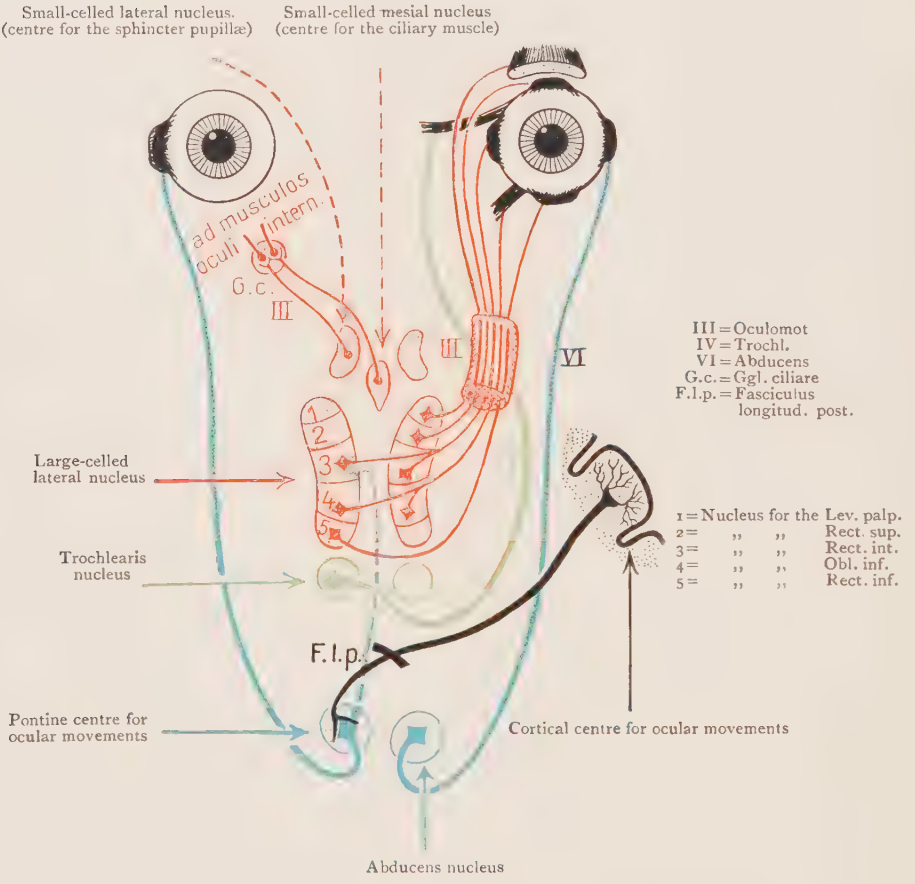


FIG. 64.—INNERVATION OF THE OCULAR MUSCLES.
III = Oculo-motor nerve.
IV = Trochlear nerve.
VI = Abducens nerve.

The fibres from (1) and (2) arise exclusively from the nucleus of the same side, those from (5) exclusively from that of the opposite side, while those from (3) and (4) arise from the nuclei of both sides.

The mesial nucleus is the centre for accommodation—*i.e.*, for the ciliary muscle, whose contraction, as is well known, loosens the *zonula* of the lens; the Westphal-Edinger nucleus, on the other hand, innervates the *sphincter*

pupillæ, and is thus the antagonist of the pupil-dilating *centrum cilio-spinale* in the lower cervical cord (*vide* p. 60). Like the fibres from the latter centre, but unlike those from the large-celled nucleus of the oculo-motor nerve, the fibres from the two small-celled nuclei of the latter do not pass directly to the muscles they supply, but connect with fresh neurons in the ciliary ganglion of the same side. These latter neurons are sympathetic, or, better, para-sympathetic, in correspondence with the smooth or "unstriped" character of the internal muscles of the eye.*

Immediately caudal to the large-celled lateral nucleus is the nucleus of the trochlear nerve, whose fibres, after undergoing a complete decussation, innervate the superior oblique muscle.

Much farther in a caudal direction—*i.e.*, in the caudal region of the pons—lies the abducens nucleus. Its fibres pass to the *rectus externus* of the same side. They are, however, connected by long collateral fibres with that part of the oculo-motor nucleus of the same side which helps in the innervation of the *rectus internus* of the opposite side. These collaterals run in the dorsal longitudinal bundle.

What now are the functions of the external ocular muscles ?

1. The *levator palpebræ* elevates the upper eyelid.

2. The *rectus internus* and *externus* rotate the eyeball round a vertical axis—*i.e.*, inwards or outwards.

3. The *rectus superior* and *inferior* rotate it round a transverse axis—*i.e.*, upwards and downwards.

4. The *obliquus superior* and *inferior* rotate the bulb round a sagittal axis; the former rolls the upper, the latter the lower, part of the circumference of the eyeball towards the nasal wall of the orbit.

The following, however, is to be noted: Only the rectus externus and internus have an uncomplicated action, the one acting exclusively as an abductor, the other exclusively as an adductor—therefore in strict antagonism the one with the other. The actions of the other four muscles of the bulb are, on the contrary, complex, the rectus superior and inferior acting, in addition to their main function, as adductors; while the two obliqui are, secondarily, abductors, and possess, further, the superior a depressing, the inferior an elevating, action on the eyeball. The statements made above, therefore, with reference to rotation about a transverse and a sagittal axis must be taken in a qualified sense. For a more detailed treatment of this subject, reference should be made to works on ophthalmology, as also for an account of the more minute diagnosis of disturbances of function in the ocular muscles (double vision, etc.).

* The ciliary ganglion belongs to the "cranial-autonomous" system, thus to the sympathetic only in a broad sense; in it takes place the passage of nervous stimuli from the white, medullary-sheathed, pre-ganglional to the grey, sheathless, post-ganglional fibres.

In directing the eyes upon a given object, however, not only do several muscles of the same eye work together, but also muscles of the one eye with those of the other, and the muscles may be associated in varying combinations according to the movements to be executed. The rectus internus, for example, is associated, in the movement of convergence, with the like-named muscle, and, in looking to the side, with the rectus externus of the other eye. That the cortical centres for ocular movements are association centres, in view of the statements made above, goes without saying. The connections between these association centres and the various subordinate parts of the oculo-motor nuclei on both sides make the mechanism as a whole one of great complexity, and one concerning which many points are still obscure. The conditions of innervation governing the apparatus for looking sideways are on the whole the simplest; the abducens nucleus, it would seem, acts also as a pontine centre for ocular movements, or possibly has, in close contiguity to it, a special pontine centre. The supranuclear tract* conducts (*vide* Fig. 64) impulses from the cortical centre in the opposite hemisphere to the ganglion cells of the abducens nucleus; the axis cylinders of the latter pass, in the first place, to the rectus externus of the same side, and, in the second place, by means of their collaterals running in the dorsal longitudinal bundle, cause contraction of the rectus internus of the other eye. The cortical centre for the movements involved in looking sideways effects, therefore, a rotation of *both* eyes towards the opposite side.

Just as fibres from the hypoglossal nucleus pass into the lower nucleus of the facial, in order to participate in the innervation of the orbicularis oris, so fibres from the oculo-motor nucleus apparently enter the upper facial nucleus, and take part in the innervation of the orbicularis palpebrarum. The grounds for this conclusion are—(1) The paresis of the orbicularis which accompanies nuclear lesions of the oculo-motor nerve; (2) the upward turning of the eyeball, which takes place simultaneously with closure of the lids (Bell's phenomenon).

With regard to the symptoms which accompany lesions of the innervation mechanism we have described above, certain definite statements may be made.

In the first place, it is readily intelligible that paralyses of isolated ocular muscles are most commonly noticed in the rectus externus and obliquus superior, each of which possesses its own exclusive nerve-supply (abducens and trochlearis respectively). Paralyses of single muscles innervated by the oculo-motor are much rarer, their occurrence presupposing extremely circumscribed lesions in the nuclear region of the nerve.

Complete oculo-motor paralysis is accompanied by falling of the upper eyelid (ptosis), which is not to be confused with the narrowing of the palpebral aperture due to failure of the sympathetic innervation of the musculus tarsalis

* That is to say, the *chief* supranuclear tract; for there is, in addition, an accessory tract, arising from the cortex of the same side.

superior (*vide* pp. 60 and 124). The globe is rotated permanently downwards and outwards (rect. ext. and obl. sup.), and, in addition, the pupil is dilated and the eye permanently adapted for distant vision (paralysis of the sphincter pupillæ and musculus ciliaris). If the abducens and trochlearis are also paralyzed, we have the condition known as *ophthalmoplegia totalis*, in which the eyes are looking straight forwards and are immobile. The small-celled nuclei for the intrinsic ocular muscles are, as is seen in Fig. 64, separated from the large-celled nuclei for the extrinsic musculature. They may escape injury when the latter are destroyed, in which case there results the condition known as *ophthalmoplegia externa ;* or, on the other hand, they may suffer destruction alone (*ophthalmoplegia interna*).

Where co-ordinated movements of several muscles are impaired, we speak of *conjugate paralyses*. Thus, the patient may be unable to look to the right or to the left, upwards or downwards, or to move the eyes convergently. In these cases the *association* centres are involved. The simplest form of these association paralyses is that presented by the abducens, whose nucleus may be regarded as functioning also as a pontine association centre. Nuclear abducens lesions are in consequence followed (with very rare exceptions, as, for instance, in encephalitis lethargica), not by simple abducens paralysis, but by inability to look towards the side of the injured nucleus. It is only when the abducens fibres are interrupted peripherally to their nucleus—*i.e.*, after the collaterals to the rectus internus nucleus of the oculo-motor have been given off—that we have an isolated paralysis of the rectus externus.

The following rules may be laid down for localization of lesions involving paralyses of the ocular muscles:

1. Unilateral paralyses of ocular muscles, whether multiple or isolated, are almost always of nuclear or infranuclear type. The supranuclear innervation of the ocular muscles is preponderatingly bilateral, the conditions resembling those described in the case of the laryngeal centre (*vide* Fig. 56 and p. 107). In general, supranuclear paralyses of the eye muscles only occur in affections involving both hemispheres, and in these only very seldom, as the cortico-nuclear fibres under consideration do not travel in compact bundles, but are much scattered in their course from the cerebral surface. Paralyses of the eye muscles cannot therefore be included in the clinical picture of pseudo-bulbar paralysis drawn above (pp. 108 and 109).

Morbid processes involving a large extent of the cortex on both sides are the most likely to cause bilateral ophthalmoplegias (meningitis).* An exception to the foregoing is afforded by ptosis. Isolated crossed paralysis of the levator palpebræ is not uncommon in unilateral cortical lesions, from which it

* In contrast with the nuclear and infranuclear paralyses of ocular muscles, in these supranuclear ophthalmoplegias reflex ocular movements may be started from the labyrinth (" caloric nystagmus," *vide infra*, p. 134).

would seem that in a good many individuals the cortical innervation of this muscle is predominantly contralateral.

2. The cortical innervation for the most important associated movement, the direction of both eyes to one side, is, broadly speaking, a contralateral one. The cortico-nuclear tract passes, as shown in Fig. 64, from a particular portion (for the localization of this centre at the foot of the second frontal convolution, *vide infra* in the section on the Cerebrum) of the cortex of one hemisphere to the abducens or pontine centre of the opposite side. This supranuclear tract

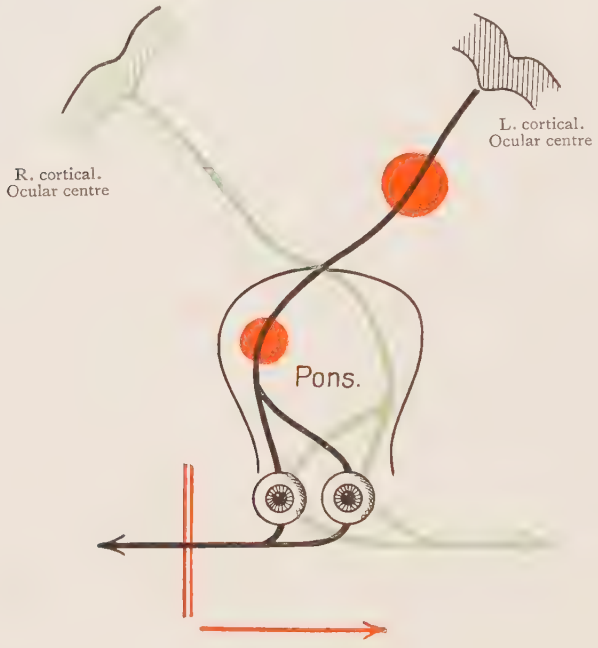


FIG. 65.—CONJUGATE DEVIATION.

The red circles represent two distinct foci (one right- and one left-sided), which may cause failure of conjugate ocular movements to the right, and consequent direction to the left (red arrow) due to the unimpeded action of antagonist muscles.

crosses the middle line at a spot, marked in our figure, which lies at the level of the anterior border of the pons. If, now, this tract is interrupted by a lesion proximal to the point of decussation, we shall have, supposing the lesion to be on the right side, inability to look to the left; if, on the other hand, the lesion is situated in the pons—thus distal to the crossing—the patient will be unable to look to the right. The unopposed action of the sound antagonists causes a deviation of both eyes—in the first case towards the right, in the second towards the left. In any case, therefore, in which this "conjugate deviation" has resulted from interruption of the cortico-nuclear tracts, the patient, if the

lesion is above the pons, looks towards the side on which his lesion is situated; if the lesion is in the pons, he looks, on the contrary, away from the side of the lesion. The rules for diagnosis of a pontine lesion have been already fully discussed. Conjugate deviation is not a lasting symptom, for, apparently, the accessory cortico-nuclear connections of the same side may gradually take over the work vicariously.

3. Nuclear paralyses of the oculo-motor are, in consequence of the great extent of the nucleus, seldom total. The sphincter pupillæ and musculus ciliaris generally remain unaffected. Paresis of the orbicularis palpebrarum points to a nuclear as distinguished from an infranuclear lesion (*vide supra*, and compare the analogous relation between the hypoglossal nucleus and the orbicularis oris). In progressive nuclear paralyses of the oculo-motorius ptosis generally first appears when the recti superior, inferior, and internus have become step by step involved, so that, as the morbid process reaches its culminating point," the curtain finally falls." A nuclear lesion of the abducens causes, as we have seen, almost always, not isolated paralysis of the rectus externus of the same side, but conjugate paralysis affecting the rectus externus on the side of the lesion, and the rectus internus of the other eye, the result being conjugate deviation towards the sound side. In this condition, it should be expressly noted, convergence is not interfered with if the oculo-motor be intact. Further, nuclear abducens paralysis is accompanied by facial paralysis of peripheral type, owing to the proximity of the "genu" of the facial as it turns round the abducens nucleus.

4. In infranuclear paralysis of the oculo-motor, the sphincter pupillæ and the ciliary muscle practically never escape, as their neurons are in the closest contiguity with those for the external ocular muscles in the oculo-motor trunk. Infranuclear abducens lesions cause isolated paralysis of the external rectus, not conjugate paralysis of that and the rectus internus of the other eye.

5. Hemiplegia alternans superior *vel* oculo-motoria, or Weber's paralysis, is characteristic of infranuclear oculo-motor affections (*vide* p. 95). The lesions are generally situated within the brain-stem (peduncular oculo-motor paralysis). They are, however, occasionally basal. In Foville's paralysis (*hemiplegia alternans abducento-facialis*) the abducens affection is generally also infranuclear.

6. The basal situation of a lesion giving rise to ophthalmoplegia is indicated, apart from the general signs of an intracranial and the special signs of a basal affection, by the simultaneous involvement of other cranial nerves (anosmia, amaurosis, trigeminal anæsthesia, facial paralysis, nerve deafness). Where we have progressive pathological processes (gummata, tumours, meningitis) in the neighbourhood of the oculo-motor emergence ptosis is usually the first symptom. Fractures of the base involve, most frequently, the

abducens, which occupies a very exposed position at the summit of the pyramid of the petrous bone (*vide* Fig. 55).

Of irritative symptoms affecting the ocular muscles, nystagmus first deserves mention. This is the name given, as is known, to rhythmic contractions of the ocular muscles, which may come on in the position of rest (*spontaneous nystagmus*), but are more frequently evoked by voluntary movements of the muscles (*intention tremor*). Horizontal nystagmus is the form most frequently observed. There are, however, vertical, rotatory, and indeed retractory forms (*vide supra*, p. 101). Nystagmus is also found as a consequence of congenital weakness of sight, or of such as comes on early, before the individual has thoroughly learnt to "fix" the eyes; further, as an occupation spasm, in coal-miners. We shall have something to say about nystagmus in the section on the Cerebellum. In the nystagmus caused by irritation of the inner ear (cold or hot douching,* acute inflammations), the afferent impulses pass by way of the connection between the nucleus of Deiters and the dorsal longitudinal bundle. Tegmental lesions in the neighbourhood of the latter may also give rise to nystagmus (brought on in this case by looking to the side on which the lesion is situated), as also may lesions in the neighbourhood of the abducens nucleus or pontine centre. Pressure in the neighbourhood of the corpora quadrigemina, under which lie the oculo-motor and trochlear nuclei (*e.g.*, by tumours in the anterior portion of the cerebellum), causes occasionally a vertical upward nystagmus, more rarely a retractory. Lesions near the abducens nucleus may also cause nystagmus, and in rare cases a conjugate deviation, which is to be regarded as an irritative symptom, the deviation being to the side of the irritated nucleus. Of much greater importance and significance is the conjugate deviation due to irritation, not to paralysis, of the cortical centre. This will, however, be dealt with later, when we come to discuss cortical localization.

The symptomatology of the pupillary reflexes will be considered in connection with affections of the optic nerve.

B. LESIONS OF THE CEREBELLUM.

The cerebellum is, from an anatomical point of view, to be regarded as a self-contained structure with well-defined boundaries. In its capsule, formed by the posterior cranial fossa and the tentorium, it represents a more or less independent annexe to the rest of the central nervous system, with which it is connected by three pairs of crura or peduncles:

* Syringing the ear with cold water causes in the healthy a nystagmus towards the opposite side. If, on the contrary, hot water is used, the movement is towards the side of the syringed ear (Barany's experiment, "caloric nystagmus"). Differences in temperature cause movements in the endolymph which have an irritative action on the nervous apparatus of the labyrinth.

1. The inferior peduncles (*crura ad medullam*).
2. The middle peduncles (*crura ad pontem, brachia pontis*).
3. The superior peduncles (*crura ad cerebrum*, s. *ad laminam quadrigeminam*, s. *brachia conjunctiva*).

The inferior and middle peduncles convey chiefly afferent or *cerebellipetal* fibres to the cerebellum, while the superior peduncles or brachia conjunctiva are composed mainly of efferent (*cerebellifugal*) fibres.

In the cerebellum itself anatomists recognize a number of lobes, which, consisting as they do of cortical and medullary substance, present, on transverse section, the characteristic dendritic appearance known as *arbor vitæ cerebelli* The largest mass of grey matter contained in the substance of the cerebellum is the *nucleus dentatus*. Other cell complexes contained in the white centre of the cerebellum are the *nucleus fastigii*, *nucleus emboliformis*, and *nucleus globosus*. Clinically, however, at any rate for the present, our division of the cerebellum is a much simpler one, as we merely have to distinguish between a mesial portion, the *vermis cerebelli*, and two lateral portions, the *hemispheres*. It is true that very promising contributions towards a more exact cerebellar localization have already been made (*vide infra*, observations on direction and tone centres in the cerebellum), yet we must, for the present, be well content if, in any given case of cerebellar affection, we are able to say with certainty in which of the above main divisions of the organ the lesion is situated.

How comes it that our knowledge is here so incomplete? How are we to explain the contrast between our knowledge of this organ and of the other parts of the central nervous system that have come under our view—the spinal cord and the brain-stem—for the investigation of whose pathological processes we have at our disposal regional diagnostic weapons of such precision that we are able to recognize and to localize correctly lesions no greater than a millimetre in extent?

In the first place, anatomical conditions play some part in the matter, for, in consequence of the enclosure of the organ between the tentoruim and the base of the skull, the injurious action of all pathological processes, whose tendency is to encroach on an already closely occupied space, extends to very large portions of the organ, the conditions here being far more favourable for the production of remote effects than is the case in other parts of the central nervous system.

While, however, anatomical conditions play their part, the comparative difficulty of localization in cerebellar lesions is much more largely to be ascribed to the relatively small physiological importance of the organ. The cerebellum is neither a motor nor a sensory organ, nor, in the human subject at any rate, has any clearly defined arrangement of centres for specific functions passed at present beyond the stage of suggestion. It acts rather as an accessory

apparatus, in close connection with the cerebro-spinal axis, but in no way vying with it in importance, exerting a modifying influence on impulses originated therein, but not originating impulses of its own. In consequence, impairment of cerebellar functions due to pathological processes may be, with comparative ease and completeness, compensated for by the vicarious action of other nerve centres, especially those of the cerebral cortex.

The cerebellum is a reflex apparatus whose function is the preservation of equilibrium in standing and walking. It is, therefore, not the seat of any independent function, but of a certain reaction to centripetal impulses.

The centripetal impulses in question result from the constantly changing positions of the body and of its parts in relation to external objects. The centrifugal impulses automatically evoked by them in the cerebellum, however, govern and modify the voluntary movements called into play by the cerebrum in such a manner as to secure the preservation of equilibrium. To express the matter in more precise terms, the cerebellum provides for the proper co-operation of various groups of muscles (*synergia*), and correctly graduates the strength of the nervous impulses passing to them (*eumetria*). It acts, in a word, as a co-ordinator and a moderator, and, by its action, insures that the centre of gravity of the body shall be in stable equilibrium while the body is standing erect, and that, in walking, the equilibrium of the body shall be maintained, and there shall be no tottering or staggering.

The afferent impulses which automatically start into action this subtle regulating mechanism are brought to the cerebellum by two routes—namely:

1. *Via* the spino-cerebellar tracts. We have here to do with a part of the impulses included under the term "deep sensibility," which come from the muscles and joints of the vertebral column and the extremities, especially the lower (*cf.* pp. 15 and 16). The great majority of the fibres of the spino-cerebellar system arise from the lumbar and dorsal cord. In the preservation of equilibrium, alike in standing and in walking, the control of the legs and trunk is of the first importance.

2. *Via* the nucleus of Deiters, whose direct connections with the cerebellum are shown in Fig. 59. The figure shows also the tract which leads from the utricle and the saccule to the nucleus in question—the vestibular tract. The vestibular nerve, as the nerve for space perceptions, conveys to the cerebellum information concerning the position of the body, especially of the head, in surrounding space. Thanks to the numerous association fibres between the nuclei of the ocular muscles and the nucleus of Deiters, the cerebellum is also, however, kept informed of the position of the eyeballs and the state of contraction of their muscles, if such an expression may be used in the case of impulses of a subconscious nature. The importance of these perceptive impulses conveyed from the ocular muscles, in the estimation of our position in space, is shown by the fact that their state of contraction varies with every

change of our position with relation to external objects, plays an important part in the estimation of distance, and so on.

We must now consider the tracts which convey efferent impulses from the cerebellum to the organs of motion, and thus act as motor limbs of the cerebellar reflex arc. It may be stated, as a preliminary, that these efferent reflex limbs are never formed by a simple neuron stretching from the cerebellum to the motor cells, but always by a chain of neurons, the most important connecting stations in which are the nucleus of Deiters, the optic thalamus, and the red nucleus of the tegmentum.

The nucleus of Deiters is thus not merely a sensory, but also a motor nucleus. We have already (p. 16) made acquaintance with the vestibulo-spinal tracts as a class of subcortico-spinal tracts which, in pyramid lesions, convey impulses to the cells of the ventral horn, so that the latter are not entirely cut off from central innervation. Under normal conditions, however, they act under the governance of the cerebellum, and regulate the supply of nervous impulses to the motor cells in the ventral horn.

In addition to the above, however, a portion of the centrifugal fibres of the nucleus of Deiters, passing in a frontal direction along the dorsal longitudinal bundle, connects with the cells of the oculo-motor, trochlear, and abducens nuclei. The cerebellum therefore provides, also, for *synergia* and *eumetria* in connection with ocular movements.

Further, the red nucleus of the tegmentum sends, as we have seen, the rubro-spinal tract (*Monakow's bundle*) to the cells of the ventral horn. As, however, a strong contingent of efferent fibres from the cerebellum passes through the superior cerebellar peduncles (*brachia conjunctiva*) to the cells of origin of this tract, they are held to play a part in the cerebellar regulation of motor impulses analogous to that of the vestibulo-spinal tracts.

The cerebellum, then, to summarize, provides, by means of the spino-cerebellar tracts and the system of the nucleus of Deiters, for the perception of the position of our body, limbs, head, and optic axes; while, by means of the vestibulo-spinal and rubro-spinal tracts, and the dorsal longitudinal bundle, it regulates the innervation of our trunk, limb, and eye muscles in such manner as to secure a continuous and harmonious maintenance of equilibrium in standing or walking.

Cerebellar ataxia, therefore, a typical disturbance of co-ordination, is by far the most important symptom of cerebellar affections.

In contrast with *posterior root ataxia*, in which all movements exhibit loss of co-ordination, cerebellar ataxia affects specially the larger movements, movements involving wide co-operation among groups of muscles, especially of the trunk and lower extremities. Single movements, such as flexion and extension of the foot, knee, and hip, abduction and adduction of the thigh,

may be correctly executed. Their static and dynamic co-ordination, however, is interfered with in such a manner as to produce a zigzag gait, like that of a drunken man, while the power of standing in an erect posture is impaired by a more or less marked tendency to stagger (*vide* Fig. 66). The separate trunk and limb muscles can no longer co-operate correctly in maintaining rest and stability simultaneously. This cerebellar *asynergia* may reveal itself also by a tendency, while walking forwards, for the head and trunk to lag behind, to fall backwards—a tendency which may so seriously disturb the equilibrium of the body as to bring about a fall; or, again, in rising from the supine position, the legs may be lifted instead of the body, and so on.

Fig. 66.—Cerebellar Ataxia.

The upper extremities are, as a rule, only very slightly affected in cerebellar ataxia; in some cases they appear to escape altogether. Some degree of uncertainty in the action of grasping with the hand is, however, observed in most cases. The upper extremities are little subject to the co-ordinating influence of the cerebellum, for the reason that they have little to do with the maintenance of equilibrium. Occasionally, however, one can prove the existence of a latent disturbance of co-ordination by a kind of artifice. The patient is told to carry out in rapid succession movements of antagonistic muscles—*e.g.*, movements of pronation and supination. If cerebellar ataxia be present, the patient will often prove unable to carry out the order, to bring about so subtle a co-operation between antagonist muscles. This abnormality is termed *adiadochokinesia* (διαδοχή = succession).

The noticeable slowing of speech (*bradylalia*) which is to be observed in

many sufferers from cerebellar affections may also be regarded as an adiadochokinetic disturbance.

The spino-cerebellar tracts form the afferent or centripetal limb of a reflex arc, regulative, not only of muscular movements, but also of muscular tone ("statotonus"). Their experimental section causes, in addition to ataxia, hypotonus in the musculature of the extremities on the affected side. It is not to be wondered at, then, that, as a rule, cerebellar ataxia is accompanied by cerebellar hypotonus.

This cerebellar hyptonous can be demonstrated, like the spinal hypotonus due to lesions of the posterior root system, by palpation, by testing with resisted movements,* and by the possibility, owing to loss of tone in antagonizing muscles, of putting the limbs in abnormal positions (*vide supra*, p. 17). While, however, as we have seen, spinal hypotonus is accompanied by impairment or abolition of reflexes, cerebellar hypotonus is independent of the condition of the reflexes. After ablation of one lateral lobe of the cerebellum in man (operation for cerebellar tumours), the reflexes, even if absent immediately after the operation, soon return, sometimes in exaggerated degree, the condition of hypotonus, on the other hand, persisting. Somewhat different, indeed, are the conditions obtaining in these "pure" cases of cerebellar lesion (comparable to those produced in animal experimentation) from those found in cerebellar diseases which encroach on space, and so cause pressure (*e.g.*, tumours). Here one finds, not infrequently, abolition of tendon reflexes, especially of the patellar and Achilles reflexes. This is, however, to be regarded as a remote effect. The increased intracranial pressure is communicated to the dural envelope of the spinal cord, and injures, as has been proved anatomically, the centripetal fibres for those reflexes at the point where the posterior roots pass through the enclosing prolongation of the dura mater. This point is, as the pathology of tabes dorsalis shows, to be regarded as a *locus minoris resistentiæ*. It has been possible in some cases to prove histologically the connection between pressure lesions of this kind and ascending degenerations of the dorsal columns of the spinal cord.

Cerebellar ataxia and hypotonus are most fully developed in diseases of the vermis, in whose grey matter it is that the spino-cerebellar tracts find their termination. In unilateral or mainly unilateral cerebellar lesions, ataxia and hypotonus may in some cases affect the trunk and extremities on the same side only as the lesion; in other cases, while not entirely homolateral, they are much more marked on the same side than on the opposite one. This also is fully explained by the anatomical conditions, for the spino-cerebellar tracts, which,

* A special test for cerebellar hypotonus is the following: If one resists an effort at flexion on the part of the patient and then suddenly ceases resistance, flexion, of extreme degree, follows, and the "rebound" which normally succeeds is either absent or is only faintly indicated, both expressions of extensor hypotonus.

with the exception of a small portion of the bundle of Gowers, undergo no decussation in the cord, traverse the cerebellum also uncrossed. The lateral cerebellar tract passes through the homolateral *corpus restiforme*, and then round the homolateral *nucleus dentatus;* the tract of Gowers, through the homolateral superior cerebellar peduncle. The termination in the cortex of the vermis takes place, it is true, for the most part after the middle line has been crossed, but so near to the latter that the termination may almost be regarded as mesial (*vide* Fig. 67).

FIG. 67.—COURSE AND TERMINATION OF THE SPINO-CEREBELLAR TRACTS (LATERAL CEREBELLAR TRACT AND TRACT OF GOWERS).

Through the efferent limbs of the reflex arc also, the cerebello-rubro-spinal and the cerebello-vestibulo-spinal tracts, each half of the cerebellum is connected with the homolateral half of the spinal cord. It is true that each cerebellar hemisphere is connected with the opposite red nucleus of the tegmentum, but this decussation is countervailed by the decussation, in the opposite sense, of Monakow's bundle (*vide* Fig. 68). The cerebello-vestibulo-spinal tract, however, undergoes no decussation in any part of its course.

Worthy of mention, but lacking at present a satisfactory physio-pathological explanation, is the fact, frequently observed, that in unilateral cerebellar affections the ataxia and hypotonus homolateral to the lesion are associated with a noticeable and regular under-estimation of the weight of an object held in the hand while the eyes are shut.

Singular disturbances of tonus and co-ordination may be made apparent by an experiment whose results point to the existence in the cerebellum of centres for controlling the direction of movements of the limbs. If a healthy individual is asked to touch a finger held up before him, first with open and then with closed eyes, he will succeed in both tests. In sufferers from cerebellar affections, however, there may be, on the side of the lesion, an inability to make the movement correctly when the eyes are shut, a missing of the objective, the direction of deviation (outwards or inwards, upwards or downwards) being found to be constant with repetition of the experiment. One is forced from this to infer the

existence of centres for "outward" and "inward," "upward" and "downward" tonus of each limb, or rather each articulation, centres which normally keep a due balance among themselves, but some of which gain the upper hand when equilibrium is destroyed by a lesion. It may, for instance, be regarded as probable that the centres for "downwards" and "outwards" tonus of the arm have the localization given in Fig. 69. Destruction of the areas concerned by a pathological process will therefore bring with it upward and inward deviation,* unless the cerebral apparatus takes on their function vicariously, which appears always to be the case if the affection lasts some time. The localization of other "direction centres" is still too problematical for fitting discussion here.

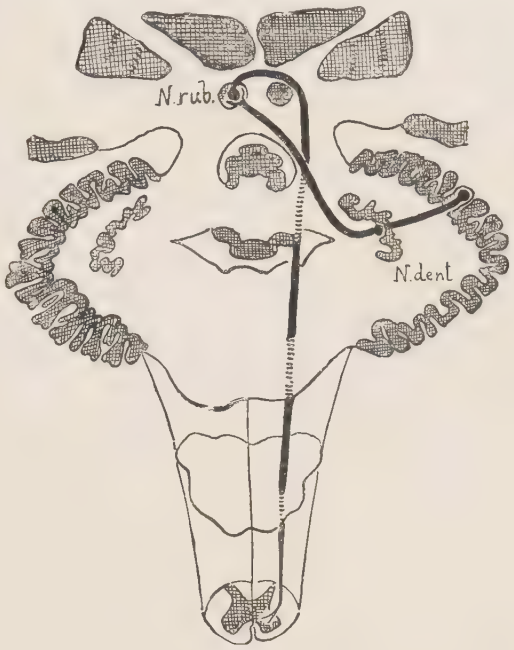


Fig. 68.—Connection of the Cerebellar Hemispheres with the Corresponding Lateral Halves of the Spinal Cord by the Cerebello-Rubro-Spinal Tract.

It has been maintained recently that the disorders of locomotion observed in cases of cerebellar disease are characterized by a tendency to stagger towards the affected side. The statement is not, however, of universal application, the opposite tendency being not seldom found.

A pronounced cerebellar hemihypotonus may lead to a true homolateral hemiparesis. The hypotonus suffices to distinguish the latter from crossed cerebral hemiparesis, which is, indeed, spastic in character Babinski's sign also is absent.

Cerebellar ataxia is the cerebellar symptom *par excellence.* Cerebellar hypotonus does not approach it in significance, for the symptoms pointing to involvement of neighbouring structures, which are often present in cerebellar affections, may obscure it altogether. This is not the case with cerebellar ataxia, which may be unmistakably present side by side with marked spastic manifestations.

If we regard cerebellar lesions from the point of view of ætiology, we shall find the possible causes numerous and varied: congenital hypoplasias and

* Irritative lesions of these areas (*e.g.*, meningeal irritation) will naturally have the reverse effect, and cause downward and outward deviation.

other defects, sclerotic contractions and atrophies, hæmorrhage and softening, injuries and abscesses, tumours, gummata, and tuberculous deposits. It is clear that, of all these affections, the total and partial atrophies and aplasias will give the most typical pictures of cerebellar impairment, seeing that here the picture is not veiled by irritative phenomena and remote effects. If all those cases are carefully eliminated in which there are present other affections of the central nervous system in addition to the pure cerebellar lesion, we find that, on the one hand, partial lesions, even when they involve the destruction of a whole cerebellar hemisphere, may remain latent; while, on the other hand, lesions involving both hemispheres lead to nothing further than pure cerebellar ataxia and—probably in every case—hypotonus, though, unfortunately, in the earlier observations, attention was not paid to this latter point.

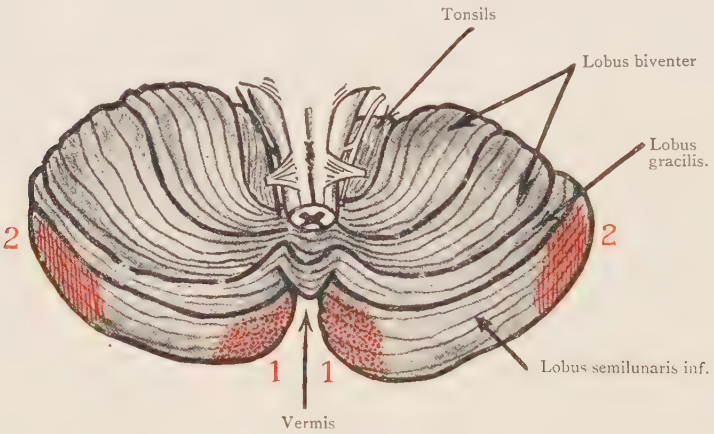


FIG. 69.—DIRECTION CENTRES IN THE CEREBELLUM.

1. Tonus centre for " downward " movements of the arm.
2. Tonus centre for " outward " movements of the arm.

None of the other symptoms, therefore, which play a part in the diagnosis of cerebellar affections, and which we shall now proceed to consider in detail, are to be regarded as pathognomonic components of the cerebellar symptom complex. They may all be evoked by non-cerebellar lesions. If cerebellar ataxia (whether alone or associated with hypotonus) be absent, we are not entitled to make a positive diagnosis of cerebellar disease. But not even these two symptoms themselves can be regarded as pathognomonic, for lesions of the cerebellifugal or cerebellipetal tracts outside the cerebellum itself may put its regulating function out of action, whether the lesion be in the mid-brain, in the pons, in the medulla, or even in the spinal cord (*cf.* Friedreich's disease). In the case of a reflex apparatus it may, under certain circumstances, amount to the same thing, whether the centripetal components, the centre, or the centrifugal components be the seat of interruption.

That vertigo, which, next to ataxic or ataxic-hypotonic disturbances, is by far the most frequently occurring symptom of cerebellar disease, is not a true cerebellar phenomenon, but a sign of disturbance of the vestibular system, is shown both by clinical observation and by experiment. The so-called "cerebellar vertigo" is, in character, very closely allied to labyrinthine vertigo —one might say, indeed, identical with it. Even when it occurs in diseases of purely intracerebellar situation, it is to be regarded as an irritative symptom, to be referred to the vestibulo-cerebellar tracts which traverse the cerebellum and enter its cortical grey matter (*vide* Fig. 57). Cerebellar vertigo is, like labyrinthine, a so-called "systematic" vertigo, a rotatory vertigo, and, as such, to be distinguished from the "asystematic" form, the diffuse disturbance of perceptions relating to position in space, which may be present in the most varied functional nervous conditions, in abnormal conditions of the cerebral circulation, in sea-sickness, etc. The sensations here—*i.e.*, in cerebellar vertigo —are of rotatory displacements in a particular direction between the patient and external objects. These sensations, however, lead, reflexly or by irradiation, to intense discomfort and feelings of malaise, which may even go so far as to cause vomiting. We would explain this phenomenon as follows:

The labyrinth performs its functions in reference to the sense of position in space in the following manner: The hydrostatic conditions present in the three semicircular canals, which lie in the three chief planes, lead to the development of nervous impulses in the vestibularis, and these pass to the cerebellum by way of the vestibular nucleus and the nucleus of Deiters. If, now, there is a contradiction between the stimuli received from the vestibular apparatus and the actual position of the body in space, the messages received from the muscles and joints, eyes, etc., will be, so to speak, "given the lie," and from this incongruence result the vertiginous imaginary movements, and indirectly their painful accompaniments.

True vestibular rotatory vertigo* may therefore be observed under the following conditions:

1. In affections of the labyrinth—*e.g.*, hæmorrhages therein (Ménière's disease in the narrower sense), primary labyrinthitis (so-called Voltolini's disease), etc.; occasionally, too, in middle-ear affections, as a remote effect.

2. In lesions of the vestibular trunk and the vestibular nucleus (basal hæmorrhages, tumours, hæmorrhages in the pons, gummata, etc.).

3. In intracerebellar affections.

* Vestibular rotatory vertigo may be brought about experimentally by means of the rotating chair or by applying the galvanic current. On passing the latter transversely from temple to temple the reaction to the consequent feeling of vertigo shows itself in a sound individual by an inclination of the head to the side of the anode. Destruction of both labyrinths or vestibulares abolishes this phenomenon completely; if the destruction is unilateral, the head inclines always to the affected side, whether anode or kathode be applied there (so-called "vertige voltaique").

The differential diagnosis between these conditions will, as a rule, offer no difficulties, if careful consideration is given to all the circumstances of the case.

In 1 and 2 we have, as a rule, accompanying auditory disturbances, either paralytic or irritative (hardness of hearing, deafness, tinnitus). Affections of the middle ear are not difficult to recognize. Further, it should be noted that, in labyrinth lesions, the onset and direction of imagined movements of external objects is often influenced by the position of the head, which, in cerebellar lesions, is hardly ever the case. Lesions affecting the peripheral vestibular neuron, between the vestibular nucleus and the labyrinth, exhibit, as a rule, in addition to the signs of vestibular disturbance, the distinguishing characters of basal or pontine lesions already enumerated. If, however, we have to do with new growths, which, in the posterior fossa, have, for the reasons stated, a marked tendency to produce remote effects, these distinguishing characters are of much less value, for cerebellar tumours injure pontine and basal structures, just as, conversely, tumours of the trunk of the acusticus,* and in the pons, impair the functions of the cerebellum.

Occasionally, in affections of the posterior cranial fossa, which increase intracranial pressure by encroachment on space, attacks of vertigo of the cerebellar or vestibular form may come on. The vertiginous paroxysms are of extreme severity, and are accompanied by symptoms of marked violence (extreme ataxia, headache, excessive vomiting, tinnitus, syncope, and nystagmus).

Nystagmus is, however, a frequently observed phenomenon, quite apart from cerebellar attacks due to disease of that organ. It will therefore be fitting to discuss here in greater detail the nature of this phenomenon, both as regards its physiological basis and its clinical significance. The three semicircular canals of the labyrinth may be experimentally irritated by the methods to be described. Here the so-called *Flouren's law* comes into operation. The irritation of a particular semicircular canal is followed by ocular movements in the plane of that canal; thus a horizontal nystagmus follows irritation of the horizontal or lateral canal, a vertical that of the vertical or posterior canal, and a rotary that of the superior or anterior canal, the orientation of which is in the frontal plane. Experimental physiology has shown further that the direction of reactive ocular movements (*ampullofugal* or *ampullopetal*) corresponds to that of the current set up in the endolymph of the canals. This is known as *Ewald's law*.

This lymphokinesis is the appropriate stimulus for the vestibular nerve endings. The stimulus travels first to the nucleus of Deiters, and then, in

* We have here chiefly to do with the so-called "tumours of the cerebello-pontine angle," which spring from the nerve sheaths of the acusticus and facialis at their point of emergence (chiefly fibromata).

correspondence with the neuron connections of that nucleus, (1) to the cerebellum (tractus vestibulo-cerebellares); (2) to the spinal motor centres (tractus vestibulo-spinales); and (3) to the nuclei for the ocular muscles, especially the pontine centre for ocular movements (posterior longitudinal bundle, *vide* p. 81).

This last-mentioned connection is of decisive importance in the development of nystagmus, whether spontaneous or "provoked," of which latter the so-called "caloric" nystagmus is of most importance for neurological diagnosis.

As has been stated above (p. 134), the latter is evoked by douching the ear with hot or cold water. Its oscillations are made up of a slow and a rapid jerk, the former constituting the essential symptom of irritation of the centre for ocular movements, while the rapid twitch is to be regarded as a hurried reflex return of the eyeballs to their normal position (it is absent if the person under experiment is anæsthetized, in which case we have simply a conjugate deviation instead of a nystagmus); the direction of the nystagmus is always stated as that of the quicker movement, which, bearing in mind Ewald's law, is entirely illogical. In a healthy individual douching with cold water causes a nystagmus towards the opposite side, while, if hot water be employed, the deviation is towards the side on which the douching is carried out.

This douching (Barany's experiment) sets up a movement in the endolymph of the labyrinth; the prominence of the lateral semicircular canal is cooled by the cold water, and the fluid contained therein undergoes a condensation which brings about a movement of endolymph from the ampulla towards the cooled part. There is thus brought about, in agreement with the foregoing statements, a slow movement of the eyes towards the side of the douched ear which alternates with a rapid movement of re-establishment in the opposite direction. If the physical conditions of the experiment are reversed by douching with hot water there is set up, by the heating of the prominence of the lateral semicircular canal and the consequent expansion of its endolymph, an ampullopetal lymphokinesis resulting in a slow movement of the eyes towards the opposite side and a homolateral nystagmus. Another method for producing experimental nystagmus by provoking a lymphokinesis is the "revolving chair test," in which the person under experiment is submitted to some ten revolutions about his long axis and then held still. In lesions of the vestibular apparatus this spinning nystagmus fails to be produced (*vide* textbooks on otology). A further method, the so-called galvanic nystagmus, is of little diagnostic value, as it can be brought on after extirpation of the labyrinth, and in spite of failure to produce the "caloric" form. (Stimulation of the left ear with 10 to 25 milliamperes causes a nystagmus to the left if the negative, to the right if the positive, pole be used.) A fourth method, applicable in the human subject, is the so-called "pneumatic" method, used in practice only by otologists, since a necessary condition is the presence of certain aural lesions—

perforation of the membrana tympani and bulging of the labyrinth wall, but with an intact labyrinth. If the air in the external auditory meatus is compressed by means of the ordinary rubber bulb apparatus, a nystagmus towards the irritated side comes on; if, on the other hand, the air is rarefied, the nystagmus is towards the opposite side.

Certain phenomena regularly accompanying experimental nystagmus are to be explained by the above-described connection between the nucleus of Deiters and the spinal motor nuclei (tractus vestibulo-spinalis). If, for instance,

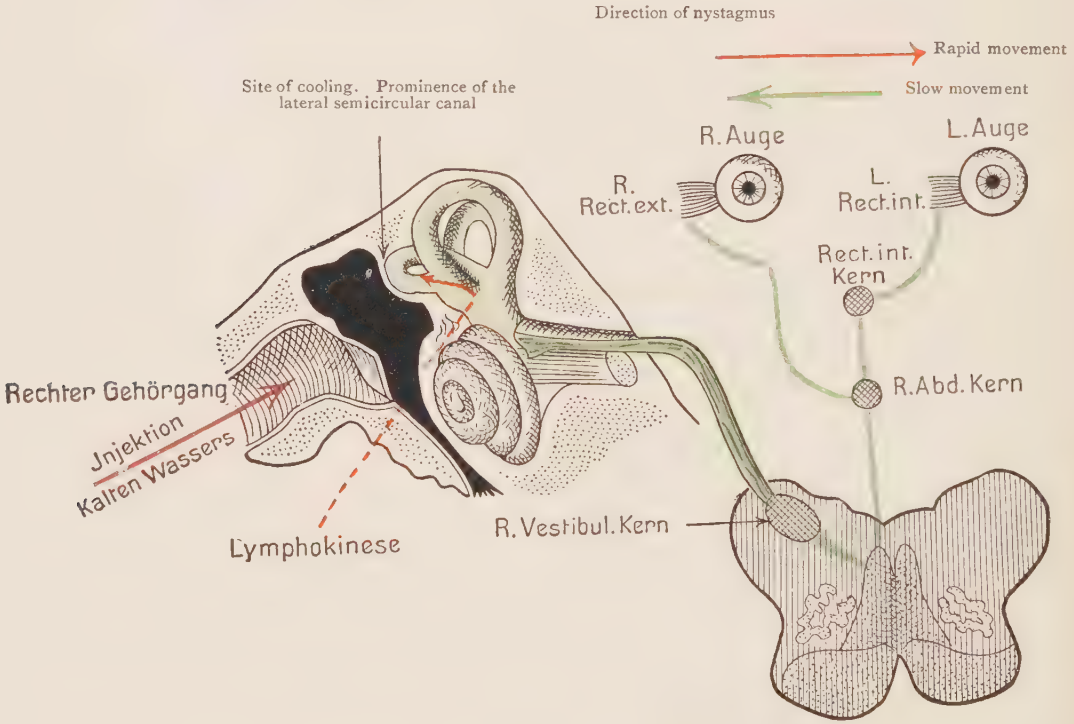


FIG. 70.—DIAGRAM IN ILLUSTRATION OF CALORIC NYSTAGMUS.

Barany's experiment is carried out on a patient standing erect with both arms held out forwards and parallel with each other, there will be observed, (1) as the nystagmus comes on, a leaning of the trunk to the side towards which the slower movement of the nystagmus cycle is directed, with tendency to fall; (2) a lateral deviation of the arms in the same direction.

Severe destructive lesions of the labyrinth and the vestibular nerve inhibit caloric nystagmus, lesser lesions diminish its intensity. Cerebellar lesions, on the other hand, hardly affect it. They do, however, influence the accompanying phenomenon described above, inasmuch as, with a unilateral

cerebellar lesion, only the contralateral arm undergoes lateral deviation. Spontaneous cerebellar nystagmus is therefore to be regarded as a long-distance effect. In those cases in which it comes on suddenly and subsequently disappears (*e.g.*, in cerebellar hæmorrhages), it appears to be a result of the condition known as "diaschisis," a kind of passive shock condition. The absence of stimuli which normally travel along nerve tracts now destroyed causes a paresis of anatomically intact areas whose activity is dependent on those stimuli. This "diaschisis" effect is essentially transitory. The sudden inhibition of cerebellar influence on the nucleus of Deiters and its connections may, in this way, temporarily throw out of order conjugate ocular movements. In pure uni- or bilateral non-development of the cerebellum nystagmus is, however, not present. When, on the other hand, it occurs as a permanent symptom of cerebellar tumours, we have to do with a pressure effect on the nucleus of Deiters, or on the posterior longitudinal bundle, which, from its dorsal position in the tegmentum, close under the vermis cerebelli, is specially exposed to pressure. This form of nystagmus seldom comes on when the patient is looking straight forwards; it is usually started by lateral ocular movements, and in such case is usually most marked when the patient looks towards the side on which his lesion is situated. In new growths of the cerebellum the phenomenon is occasionally not noticeable until the patient is placed on his side, and, moreover, on the side opposite to that of the tumour. Generally the movement of both eyes is very markedly "conjugate"; occasionally, however, a wider movement is seen on the affected side. Especially calling for mention is the vertical nystagmus which constitutes an early symptom of pressure on the corpora quadrigemina, which may be caused by a tumour in the anterior portion of the cerebellum.

Among other symptoms due to the action of cerebellar lesions on neighbouring structures may be mentioned paralysis of the abducens—*i.e.*, of associated movements of the eyeball towards the affected side—which later, in consequence of the unopposed action of sound antagonists, leads to conjugate deviation of the eyes towards the sound side. In early stages of cerebellar tumours, also in cerebellar hæmorrhages, etc., there may, on the contrary, be occasionally observed a conjugate deviation to the diseased side. In such cases we are dealing, not with a paralytic, but an irritative symptom. Such an irritative conjugate deviation may also be produced experimentally. If, for instance, we apply Barany's test to a lightly anæsthetized patient, we obtain, not a nystagmus, but a conjugate deviation towards the irritated side. The abolition of consciousness apparently inhibits the automatic return of the eyeball to its normal position, a movement which constitutes an essential element of the nystagmus cycle. Paralysis of the trochlear nerve is a fairly regular symptom of cerebellar lesions which have their seat in the most frontal portions of the vermis. Often, too, in such cases, the oculo-motor branches to the external

muscles of the eye are affected; the intrinsic muscles remain almost always intact.

Irritative and paralytic symptoms in the domain of the cranial nerves V. to XII. are, of course, chiefly to be looked for in affections which encroach on space, and, as a rule, come on earlier in extracerebellar than in intracerebellar affections. Disturbances of phonation, respiration, deglutition, and articulation may present a clinical picture closely resembling that of bulbar paralysis. Sudden death through pressure on the medulla has been repeatedly recorded. Hemiplegia may be present on the side of the lesion or on the opposite side, according as the pyramids are subjected to pressure after or before their decussation.

Forced positions and forced movements (rotation of the head or trunk to one side or the other, turning of the body about its long axis) are specially noticed in irritative affections (new growths, hæmorrhages), when they involve the superior or middle cerebellar peduncles. No rules can be laid down as to the side towards which these twistings and rotations take place, and their physio-pathological basis is, at present, equally obscure. The latter statement applies fully, also, to certain choreiform-athetotic movements affecting the extremities of one side, which may be connected with affections of the superior cerebellar peduncle of the same side, and to the so-called Magendie's squint (*skew deviation or vertical divergence*), which, coming on in association with conjugate deviation, is said to indicate a lesion of the middle peduncle. In lesions of the tract connecting the cerebellum with the frontal lobes of the cerebrum, there would appear to result a symptom seldom found in connection with cerebellar foci, the so-called "cerebellar catalepsy." The name is not altogether a happy one, seeing that the phenomenon occurs also in affections of the cerebrum—*e.g.*, in "motor" and "ideo-motor" apraxia (*vide* Chapter V.). The patient is able to maintain the same attitude for minutes together without any appearance of fatigue, and with the immobility of a lay figure (*e.g.*, lying on the back with thighs flexed and abducted, knees slightly bent, and feet rotated outwards, a position which a normal individual is unable to maintain without a constant quivering of the legs).

Finally, we would draw attention to a number of symptoms which, in cases of increased intracranial pressure due to neoplasm, justify the inference that the lesion is in the cerebellum, or at any rate in the posterior cranial fossa:

1. The headache which afflicts patients with cerebellar tumours is characterized by its extraordinary persistence and intensity. This is a consequence of the tight stretching of the tentorium cerebelli, which is rich in sensory fibres. The pain is referred chiefly to the occiput and the cervical region, from which it may radiate as far as the upper part of the back. It is also projected forwards to the forehead. While, however, this frontal pain is diffused, the occipito-nuchal is generally most acute on the side of the lesion. It is often associated with more or less retraction of the head.

2. There is often local sensitiveness to pressure or percussion in the occipital region, and occasionally a characteristic pain is caused by upward pressure on the apex of the mastoid. In both cases the pain is felt on the side of the lesion.

3. The condition of the fundus known as "choked disc" comes on very rapidly and in a very marked form, and generally on both sides. Not infrequently it leads rapidly to blindness.

4. If lumbar puncture be resorted to, pressure falls rapidly, and the flow of cerebro-spinal fluid very soon ceases. Apparently the tumour suddenly presses the medulla into the occipital foramen, and so blocks the communication between the intracranial and the spinal fluid. This involves such great danger for the patient (cases of sudden death during the procedure have been recorded) that, if a neoplasm is thought to be situated in or close to the cerebellum, the use of this diagnostic method is urgently contra-indicated.

C. LESIONS OF THE CEREBRUM, BASAL GANGLIA, AND HYPOPHYSIS.

CHAPTER I.

ANATOMICO-PHYSIOLOGICAL INTRODUCTION.

THE organ for conscious psychic phenomena is the cerebral cortex. It does not perform its tasks diffusely as a physiological unit, but to different parts of its area are assigned different functions. Both the efferent centrifugal tracts, which carry impulses from it, and the afferent centripetal, which convey stimuli to it, find their origin and termination respectively in topographically defined areas of the surface of the hemispheres—the so-called "cortical areas." While Figs. 71 and 72 give the anatomical divisions of the cortex cerebri, the fissures and convolutions, the physiological divisions, the above-named cortical areas, are represented in Figs. 73 and 74.

The cortical areas may be divided into—

1. Motor areas. From these arise the cortico-nuclear tracts which innervate our whole voluntary musculature.

2. Sensory areas. In these end the various neuron chains which serve as channels for the conduction of superficial and "deep" sensory impulses. Certain ganglion cells of these sensory cortical areas, however, not merely receive and bring into relation with our consciousness sensory stimuli, but also, in a sense, store them up, producing memory pictures. The memory pictures of the muscular sense—*kinæsthetic* memory pictures—facilitate the performance of movements whose previous performance is recollected. For this reason the

kinæsthetic and the motor areas coincide. When a kinæsthetic perception reaches our consciousness, it is thus able to translate itself at once into the

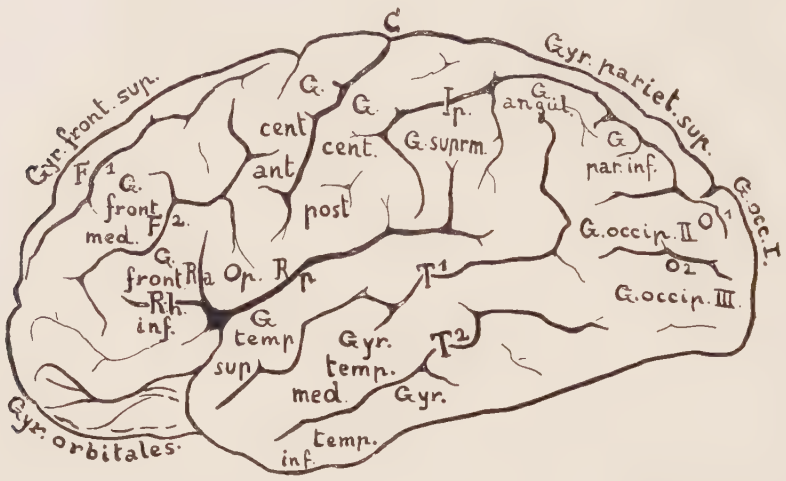


FIG. 71.—ANATOMICAL DIVISIONS OF THE LATERAL CEREBRAL SURFACE.

F^1, F^2=First and second frontal fissures; C=Central fissure; Ip.=Interparietal fissure; O^1, O^2=First and second occipital fissures; T^1, T^2=First and second temporal fissures; R.h., R.a., R.p.=Ramus horizontalis, ascendens, and posterior fissuræ Sylvii; G.=Gyrus.

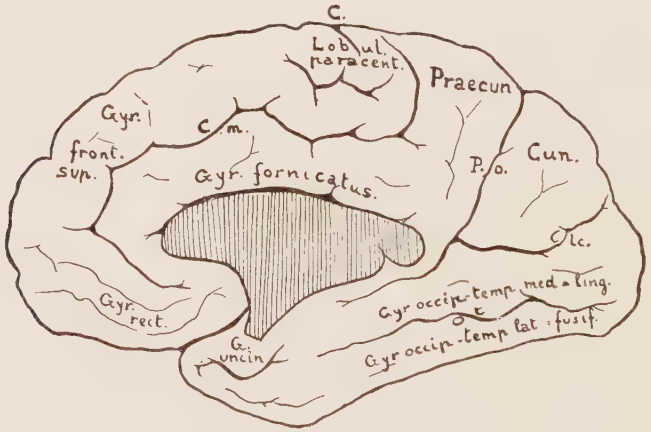


FIG. 72.—ANATOMICAL DIVISIONS OF THE MESIAL CEREBRAL SURFACE.

C.=Central fissure; C.m.=Sulcus calloso-marginalis; P.o.=Parieto-occipital fissure; Clc.=Calcarine fissure; O.t.=Occipito-temporal fissure.

appropriate movement. The cortical areas for the superficial sensibility also coincide, in the main, with the cortical motor zone (called, therefore, the "sensori-motor zone"). They, however, somewhat overlap it.

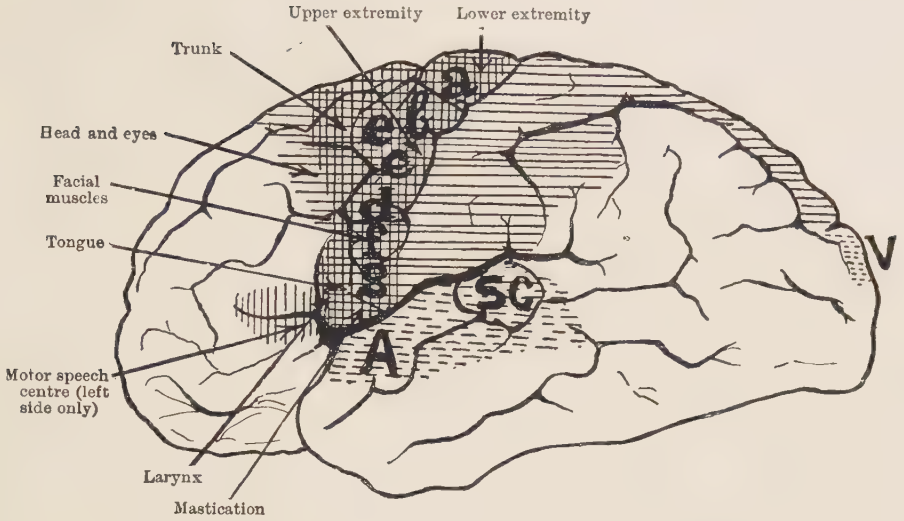


FIG. 73.—MOTOR, SENSORY, AND SPECIAL SENSE AREAS IN THE CORTEX.

= Motor area. = Sensory area. = Special sense area.

A.=Auditory; V.=Visual cortical area; S.C.=Sensory speech centre (left side only); *a*=Foot centre; *b*=Elbow centre; *c*=Hand; *d*=Fingers; *e*=Shoulder; *f*=Upper facial; *g*=Lower facial.

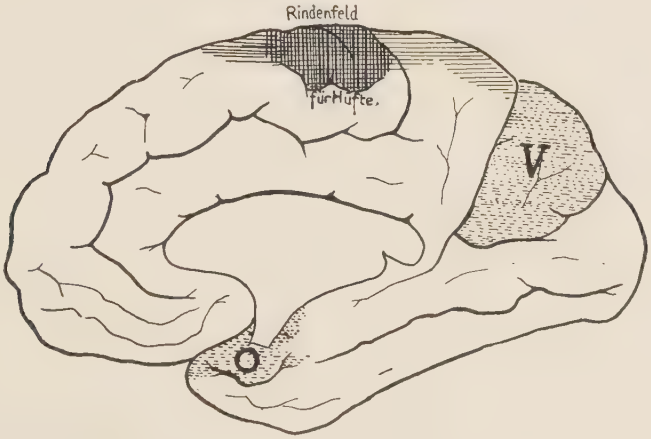


FIG. 74.—MOTOR, SENSORY, AND SPECIAL SENSE AREAS IN THE MESIAL CORTEX.

= Motor area.
= Sensory area.
= Special sense area.

V=visual, O.=olfactory area.

3. Cortical areas for special sense. These are the terminal areas of the visual, auditory, olfactory, and gustatory tracts, and the "storing-up place" for memory pictures connected with these special senses.

The simpler memory pictures (for tactile impressions, tones, colours, forms, etc.) are subordinate to the more complex (for the appearance of objects, for the spoken, written, or printed word, etc.). Association fibres which connect these different higher centres of perception with each other raise the percepts into concepts, so that, for instance, the reading of a particular word leads to the understanding of its meaning, and, at the same time, also awakens a concept of the object denoted by the word; or, again, so that the auditory perception of the ringing of a bell awakens in us, not merely the concept of the "object," bell, but also brings before us the word "bell," and at once places at our disposal the kinæsthetic memory pictures required for the pronunciation and writing of the word. Associative connections between the various concepts built up of percepts provide, finally, the foundation for our higher psychical functions ("*Nihil est in intellectu quod non fuerit in sensu*"). Concerning the manner in which this is done, however, we have at present no full anatomico-physiological knowledge.

In our Figs. 73 and 74 the various motor, sensory, and special sense areas are distinguished from each other by differences of shading. The physiological significance of the portions of the cortex left unshaded is still obscure. The present state of our knowledge, however, justifies us in claiming for the frontal portion of those areas the formation of concepts and their synthesis into intellectual personality, while the unshaded temporo-occipito-parietal area contains centres which are intercalated in the association tracts between the spheres of common sensation, sight, and hearing. The cortical centre for taste is not represented in our figures, for the reason that its localization is still matter of dispute, and is, in fact, at present the subject of mere hypothesis. Some place it in the inferior frontal convolution, others allot it a common area with the olfactory centre; in the present state of our knowledge no decisive conclusion can be formed.

Among the association tracts which connect separate areas of the cerebral cortex with one another, the so-called "commissural" tracts take a special place, inasmuch as they co-ordinate the action of symmetrically placed areas of the two hemispheres. The largest commissural complex is the corpus callosum; the anterior commissure, however, also forms an important transverse connection.

Among the neuron systems which, apart from the cortico-cortical connections, enter into relation with the cerebral cortex, the following deserve mention:

1. **Corticofugal.**—(1) *Cortico-Nuclear—i.e.*, the supra-nuclear fibres of the motor cranial and spinal nerves. The former=the cortico-bulbar, the latter =the cortico-spinal tracts.

(2) *Cortico-Subcortical.*—These fibres pass, in part directly, in part through intercalated centres, to the subcortical centres, the red nucleus of the tegmentum, the roof of the mid-brain, the cerebellum, etc. Their connecting neuron systems are, among others, the subcortico-spinal tracts (rubro-spinal, tecto-spinal, vestibulo-spinal), whose significance as accessory motor tracts we have discussed on pp. 4 and 5.

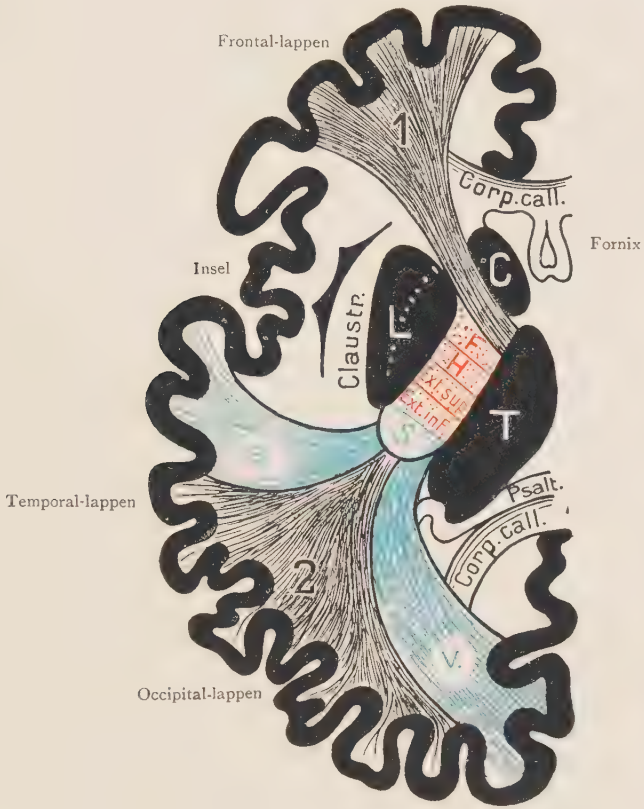


FIG. 75.—INTERNAL CAPSULE AND CORONA RADIATA.

T.=Thalamus; L.=Lenticulate nucleus; C.=Caudate nucleus; F.=Supranuclear tract for the facial nerve; H.=Supranuclear tract for hypoglossal; A.=Supranuclear tract for arm muscles; B.=Supranuclear tract for leg muscles; S.=Sensory tracts (thalamo-cortical tracts); *a*=Auditory tracts to the temporal lobe; *v*=Visual tract to the occipital lobe (tract of Gratiolet); 1=Fronto-pontine tract and corona radiata; 2=Occipito-temporo-pontine tract and corona radiata.

2. **Corticopetal.**—These are the terminal links of the neuron chains for common sensation and special sense. Their cells of origin lie in the subcortical centres—in the thalamus, for instance, for the tracts from the sensory, spinal, and cranial nerves; in the posterior quadrigeminal body and the mesial geniculate body, for the auditory tract; in the thalamus and the anterior quadrigeminal body, for the visual tract.

The arrangement of the great majority of the corticofugal and corticopetal tracts is a very characteristic one. As (1) most of the corticopetal neuron complexes come from the thalamus, and (2) the cortico-nuclear tracts, with a contingent of the cortico-subcortical, enter the internal capsule (which is enclosed between the thalamus and the corpus striatum), there is a radial convergence of fibres from a great part of the cortex towards these two centrally situated structures. It is in this manner that the so-called *corona radiata* is formed.

That part of the corona radiata which merges itself in the thalamus forms the "stalks" of the thalamus. The masses of fibres, however, which go to

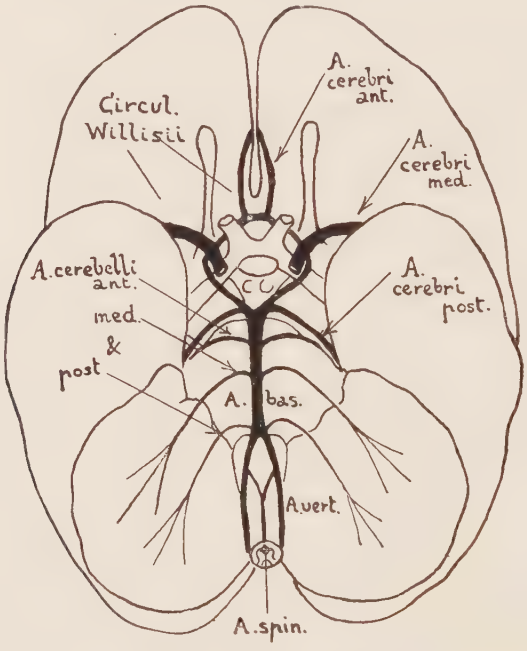


FIG. 76.—THE CHIEF ARTERIAL TRUNKS SUPPLYING THE BRAIN.

form the internal capsule are so arranged that in a horizontal section we may distinguish in the capsule an anterior and a posterior "limb," and a genu or "knee." As Fig. 75 shows, the various fibre complexes of the internal capsule, differing from each other in place of origin and in function, are arranged in a definite manner. At the "knee" lie the supranuclear facial tracts, while behind them are ranged, in order, the pyramid divisions for the hypoglossal and for the upper and lower extremities. The posterior portion of the posterior limb is occupied by the sensory tracts, and as at this point the two chief special sense tracts, the visual and the auditory, branch off for the temporal and occipital cortex, French anatomists have bestowed upon it the very appropriate

name *carrefour sensitif*. The figure requires no further detailed explanation. We would only point out, in addition to the foregoing, that the fronto-pontine and the occipito-temporo-pontine tracts (Fig. 44) contain cortico-subcortical fibres which originate in the cortical areas named, and pass thence to the pons, having their terminal ramification among certain groups of ganglion cells (the so-called "pontine nuclei"), which are scattered between the pontine fibres. The pontine nuclei, however, send their axons to the cerebellum. In this manner is established a control of the cerebellum by portions of the cerebral cortex, which we have already indicated as important association areas for special sense.

FIG. 77.—ARTERIAL SUPPLY TO THE CEREBRUM AND BASAL GANGLIA.

A=Area supplied by the anterior cerebral artery; B=Area supplied by the middle cerebral artery; C=Area supplied by the posterior cerebral artery; C.i.=Internal carotid; A.c.m. =Middle cerebral artery; 1=Lenticulo-optic artery; 2 and 3=Lenticulo-striate arteries; 3=Artery of cerebral hæmorrhage.

It will be appropriate here to consider briefly the distribution of blood-vessels to the brain. The fact that the great majority of focal lesions of the brain are of vascular origin, and that their situation is generally in close relation with the arterial distribution, gives the matter a very special importance. Two pairs of arteries supply the brain with blood—the internal carotids and the vertebral arteries (*cf.* Figs. 76-79).

The latter, after supplying the medulla, unite to form the basilar artery, which supplies, not only the pons, but also, by its branches the cerebellar arteries, the cerebellum. The anterior cerebellar artery supplies the dorsal, and the middle and posterior branches the ventral portions of the organ. At

the point where the crura cerebri diverge, the basilar divides into the two posterior cerebral arteries, which, after winding round the peduncles and giving off branches to the corpora quadrigemina, supply the lower portions of the

FIG. 78.—ARTERIAL SUPPLY OF THE LATERAL CEREBRAL SURFACE.
A=Area supplied by the anterior cerebral artery.
B=Area supplied by the middle cerebral artery.
C=Area supplied by the posterior cerebral artery.

temporal and occipital lobes. Each posterior cerebral artery sends, finally, to the internal carotid of the same side, a posterior communicating artery.

FIG. 79.—ARTERIAL SUPPLY OF THE MESIAL CEREBRAL SURFACE.
Blue =Area supplied by the anterior cerebral artery.
Green=Area supplied by the posterior cerebral artery.

The internal carotid divides into two branches—the anterior and middle cerebral arteries. As the two anterior cerebral arteries are united by an anastomosis (the anterior communicating artery), we have a complete arterial

ring formed round the infundibulum and the optic commissure (circle of Willis). The anterior cerebral arteries pass forwards in the great median fissure, and turn round the border of the corpus callosum. They supply the anterior portion of the frontal lobes and the mesial surface of the hemispheres as far as the parieto-occipital fissure.

Of these arteries, however, the most important from a pathological point of view is the middle cerebral (*cf.* Fig. 77). Its main trunk passes, as the Sylvian artery, to the island of Reil, where it splits up into its terminal branches. Previously, however, it gives off, at the base of the brain, perpendicular branches to the optic thalamus, corpus striatum, and internal capsule (arteria lenticulo-optica and arteriæ lenticulo-striatæ). One of the latter, which creeps along the outer surface of the lenticulate nucleus to the internal capsule, passes through the substance of the latter, and terminates in the caudate nucleus, is the so-called "artery of cerebral hæmorrhage," so named because the majority of cases of apoplexy are brought about by its rupture. These perforating basal arteries are, in contrast with the arteries of the cortex, which anastomose freely with one another, so-called "end arteries," and their embolic or thrombotic plugging is, therefore, far more likely to be followed by ischæmic degeneration of the parts supplied by them than is the case with the cortical vessels.

Further, as is shown in Fig. 78, the arteria fissuræ Sylvii gives off, in its course, branches to the lower and middle frontal convolutions, to the central convolutions, the supramarginal and angular lobes, and to the outer surface of the temporal lobe—thus, to the motor and sensory centre for speech, to by far the greater part of the senso-motor cortical area, and to the auditory centre in the cortex.

CHAPTER II.

LOCALIZING VALUE OF MOTOR AND SENSORY DISTURBANCES OF CEREBRAL ORIGIN.

It is obvious, from the anatomical principles stated in the foregoing section, that focal lesions of the cerebrum, which reveal themselves by motor and sensory disturbances, may vary much in locality.

They may be situated, for instance, either in the cerebral cortex, the corona radiata, or the internal capsule.

(*a*) Motor and Sensory Disturbances of Cortical Origin.

The motor areas in the cortex control, in virtue of the decussation of the pyramid fibres which issue from them, the muscles of the opposite side of the body. The connection of cortical centres with the corresponding muscles of the same side is quite subordinate. We have already had occasion to point out

that the muscles supplied by the upper part of the facial, the muscles of mastication and deglutition, the laryngeal muscles, the sterno-mastoid, and the upper or "respiratory" portion of the trapezius, as also the majority of the ocular muscles, share in this bilateral cortical innervation. It may be added here that the same holds good of the trunk muscles. This small category of muscles, therefore, will escape paralysis in unilateral focal lesions of the cerebrum. As, however, the great majority of muscles have a purely contralateral innervation, we are justified in giving the title "*cerebral hemiplegia*" to those forms of paralysis due to interruption of the motor impulses from a single hemisphere.

Where an interruption of the corticopetal fibres passing from the thalamus to the sensory areas in the cortex occurs, we speak, analogously, of *cerebral hemianæsthesia*.

What enables us to distinguish *motor disturbances due to cortical lesions** from those due to subcortical, especially capsular, lesions is, primarily, the circumstance that in the former case the affected muscular area is, as a rule, a small one. This is due to the great extent of the motor areas in the cortex. It will hardly ever happen that a cortical lesion will be equally and simultaneously destructive of the whole surface of the anterior central convolution, the neighbouring portions of the two upper frontal convolutions, and the paracentral lobule. The monoplegic type of supranuclear (spastic) paralysis is, therefore, a distinguishing feature of affections of this region of the cortex. Only as regards cortical paralyses occurring in childhood does this statement require some qualification, as here a hemiplegic type is not altogether unusual. Reference to Figs. 73 and 74 will facilitate localization in monoplegias of crural, brachial, facial, facio-brachial, and facio-lingual type.† The facial paralysis is, of course, limited to the lower or unilaterally innervated portion of its musculature.

Where we have to deal with tumours (the most frequent cause of cortical paralyses), the monoplegia usually takes the form of a gradually progressive paresis. In hæmorrhages we may have, at first, a hemiplegia (remote effect); it soon, however, gives place to a pure monoplegia. Transitory monoplegias, pointing to a temporary cessation of activity in a particular portion of the cortex (*fatigue symptom*), are often observed after epileptic and paralytic seizures; only lasting monoplegias are, therefore, to be regarded as pointing to true lesions of the cortex.

Spasticity is less intense in cortical than in capsular paralyses, hardly ever proceeding to late contracture; in rare cases it may be entirely absent, a fact of which no satisfactory explanation can at present be given. Bilateral lesions of the senso-motor cortical area are rare. They are most likely to depend on

* The very rare "intracortical hemiplegias" due to diffuse affections of the cortex will be dealt with later in connection with the subcortical disturbances of motility and the atypical forms of cerebral hemiplegia.

† Slight lesions of cortical motor centres may cause, instead of a paralysis or paresis, a motor apraxia (*vide infra*, Chapter V.).

a diffuse cerebral arterio-sclerosis. They naturally lead to diplegias, and if the symmetrical lesions involve the region of the operculum, with its masticatory, laryngeal, and lingual centres, and the centre for deglutition, we have the symptom complex of pseudo-bulbar paralysis (*vide* pp. 108 and 109). Tumours of one paracentral lobule are very apt, by pressure on the other, to cause paraplegia of the lower extremities.

Sensory Disturbances due to Cortical Lesions.—The topographical coincidence of the motor zone with the greater part of the sensory leads to characteristic results in many of these monoplegias. Thus we have in the paretic extremities or parts thereof a pronounced hypæsthesia, or indeed a complete anæsthesia in the sphere of common sensation (tactile and thermal sensibility and sensibility to pain), for the reason that the neurons involved have a diffuse termination in the cortical sensory area.

On the other hand, three so-called epicritical functions of a higher order than the elementary sensations are found to be more or less deeply impaired—viz., (1) in the sphere of superficial sensibility, the power to estimate differences in the intensity of tactile, painful, and thermal sensations; (2) and (3) in the spheres of both superficial and deep sensibility, the power to realize one's position in space, and to distinguish the qualities of external objects.

In regard to the first of these faculties, the comparatively coarse testing methods which we find successful in the case of peripheral, spinal, and subcortical sensory disturbances will not suffice; hairs must be used for stroking the surface, the algesimeter for estimating sensibility to pain, and so on. The fineness of perception of position in space is tested by passive movements of the limbs, and the power to localize tactile sensations by Weber's circle. As tactile properties for comparing and distinguishing external objects, their bulk, height, weight, and quality of surface are made use of, for the latter fabrics are chiefly employed—*e.g.*, silk, satin, flannel, and linen.

Sensory disturbances may follow lesions of the cortical areas emphasized in Figs. 73 and 80 (both central convolutions, the anterior portion of the superior parietal lobule, the gyrus supramarginalis, and gyrus angularis). Their topography has this peculiarity, that its boundary lines are neither axial nor radicular. In the upper extremity, for instance, each finger has, as a unit, its own sensory representation in the cortex; the same holds good, too, for the elbow and shoulder. Each of these areas apparently contains an apparatus which plays an important rôle in the faculty of orientation in space. The arrangement of sensory areas in the cortex is, in fact, according to functional and not to anatomical units. Examples of such functional units are the sole of the foot and the knee-joint.

The three fundamental functions of the sensory cortical area are not, however, equally distributed over the whole. Thus (*vide* Fig. 80):

1. Loss of the power to realize and distinguish position and relations in

space (in hand or foot) may follow damage to any part of the whole area, but is more marked the more to the front the lesion is situated, particularly if it affects the anterior central convolution.

2. The faculty of recognizing differences of form and weight is most seriously impaired by lesions of the posterior central convolution.

3. The faculty of estimating the intensity of single sensory stimuli is most obviously impaired when the lesion involves the posterior and inferior portions of the sensory cortical zone. Any lesion affecting the foot of the two central convolutions, the gyrus angularis or supramarginalis, or the superior parietal

FIG. 80.—SENSORY CORTICAL AREA.

Boundary of the cortical area for recognition of spatial relations.

Principal area of the cortical area for recognition of spatial relations.

Principal area of the cortical area for recognition of form and weight.

Cortical area for estimation of the intensity of sensory stimuli.

Principal region for stereognosis.

lobule may lead to such impairment of tactile sensibility, while more severe and extensive changes are necessary to inhibit the faculty of recognizing differences of temperature.

These statements, first made possible by utilization of the extensive experience of cortical lesions furnished by the Great War, represent both an enrichment and a refinement of our former very scanty knowledge concerning cortical sensory disturbances. In the first four editions of this compendium we were able to state only that lesions of the anterior central convolution are apt to cause sensory disturbances, chiefly in the sphere of the deep sensibility (with ataxia, impairment of orientation sense, and of the power of appreciating

form by palpation); that these disturbances reach their maximum when the centres for the extremities are affected, though similar sensory disturbances follow lesions of the upper parietal cortex and the supramarginal gyrus; and, finally, that very small lesions may be without disturbances of sensation.

A staggering gait due to ataxia of the muscles of the trunk, so-called cerebral ataxia—calling to mind cerebellar ataxia—is found in affections of the superior frontal convolution.

A typical cortical disturbance of sensation of mnestic-associative type is astereognosis, the inability to recognize objects by palpation in spite of unimpaired perceptions in the spheres of touch, movement, and orientation (*vide* note, p. 15).

Astereognosis is not, it must be admitted, an entirely unitary conception There are, for instance, side by side with those patients who, in spite of intact common sensation, are unable to give an account of the form, consistence, smoothness, or roughness of objects, others who can do all this, but are unable to recognize the essential character of objects, and thus cannot give them their names or state their purpose or method of use; so that, for instance, when a pencil is fingered with the eyes closed, all that the patient can say is, "It is round and has a point at one end"; in the case of a key, "It is cold and seems to be of metal; it is long and has something on one side of it." In the former case the primary "tactile identification" is impaired, in the latter the secondary.

Astereognosis is generally a focal symptom, and in that case points to a situation of the lesion in the middle third of the posterior central convolution of the opposite side. With it there is often found a coincident affection of the neighbouring portions of the parietal lobe, particularly of the supramarginal gyrus. It has been proved, however, that astereognosis may develop without any lesion of these parts of the cortex. Occasionally the cortical lesion is a diffuse one (*e.g.*, in progressive paralysis), especially in cases which point to impairment of the "secondary tactile identification."

Irritative symptoms play as important a part in affections of the sensomotor area as those betokening impairment of function. They may be observed pure, or in association with symptoms of paretic monoplegia.

In this connection we must place in the front rank of importance *irritative symptoms affecting the motor sphere.*

The name "cerebral monospasm" is given to certain tonic-clonic spasms which affect a hitherto normal or an already paretic muscular area (facial, arm, hand, etc.), leaving behind them a permanent paresis or a permanent exacerbation of one already existing.

Attacks of Jacksonian or cortical epilepsy point to a higher degree of cortical irritation. These attacks generally begin as monospasms, which are at first, as a rule, of tonic type. The spasm may, however, be clonic in character

ab initio, especially when the attack commences in the sphere of the lower facial. In these cases the spasms are not confined to the lower half of the face, as in an ordinary cortical monospasm, but affect the arm, and finally the leg, of the same side. If the attack commences in the arm, it generally, when the whole arm has become involved, spreads first to the face and then to the leg of the same side. The succession leg, arm, face is denominated the "crural" type. It is important to observe carefully in which muscles the first spasms of all, the so-called "motor aura," come on, for this "*signal*" symptom is of determining weight in the diagnosis of the situation of the cortical lesion. If, for instance, the attack begins in the fingers of the right hand (the contractions may at first affect the thumb alone), the focus (most frequently a neoplasm, but also, possibly, a cicatrix, a foreign body, a small abscess, or a circumscribed meningitis) will be situated in the left posterior central convolution, at the point marked *d* in Fig. 73. If the spasm commences at the left angle of the mouth, the lesion is situated on the right side at *g*. A lesion here "irritates" the neighbouring pyramid cells, and the irritation spreads gradually over the contiguous areas of the cortex, like waves produced on the surface of water by a stone. The muscles will be involved in an order corresponding to the relative positions of their cortical centres. In severe cases the Jacksonian spasms may pass over also to the opposite limbs, and may be accompanied by loss of consciousness. A very intense irritation may spread to the motor area of the opposite hemisphere by way of the commissural tracts, and, further, may deaden the higher psychic centres, especially those in the cortex of the frontal lobe. The bilaterally innervated muscles (frontal, palpebral, masticatory, sterno-mastoid, etc.) are, in these cases, affected simultaneously on both sides.

Unless the diagnosis is supported by the presence of other symptoms—symptoms of cerebral pressure, such as slowing of the pulse, obstinate headache, choked disc, cerebral vomiting, etc.—or unless a history of cranial trauma helps to confirm it, cortico-epileptic attacks do not enable one to infer with absolute certainty the presence of a lesion in the cortical motor areas. For, in the first place, there is a form of genuine epilepsy, which presents the typical Jacksonian symptomatology, the so-called "*idiopathic hemiepilepsy*"—it is true that more recent experiments and the results of surgical procedures have shown that, as a matter of fact, alterations in the region of the cortex underlie this form (*cysts, encephalitis*); and, in the second, intoxications and auto-intoxications (*alcoholism, absinthism, uræmia*, etc.) may evoke Jacksonian attacks by chemical irritation of the motor cortex. All these conditions may also lead to transitory monoplegic or hemiplegic pareses. Only where the latter are lasting in character do they furnish a fully valid argument for the organic nature of a cortical irritation; the same applies also to those cases in which Jacksonian epilepsy comes on in *already* paretic muscles.

Irritation of one particular portion of the cortex, *the base of the second*

frontal convolution, the area marked "*Head and eyes*" in Fig. 73, reveals itself by a phenomenon which has already been discussed in the section on the innervation of the eye muscles—viz., *Conjugate Deviation.* From the statements there made (pp. 129, 130, 133) it appeared that, in contrast with other movements of the eyes, the mechanism for conjugate movement to one side or the other is, in the main, unilaterally innervated. The supranuclear tract which leads to the abducens centre in the pons, whose function it is to set in action simultaneously the rectus externus of its own side and the rectus internus of the other, takes its origin in a cortical centre in the opposite hemisphere. A paralyzing lesion of this centre has the same effect as one involving the supranuclear tracts issuing from it at any point before their decussation at the anterior border of the pons—*i.e.*, it renders it impossible to look towards the opposite side, and thus brings about, by unopposed action of antagonists, a conjugate deviation towards the side of the lesion (*cf.* Fig. 65). If now, on the

FIG. 81.—CONJUGATE DEVIATION OF HEAD AND EYES WITH LEFT-SIDED FACIAL SPASM AT THE ONSET OF A JACKSONIAN ATTACK (RIGHT-SIDED CEREBRAL TUMOUR).

other hand, the cortical centre in question be irritated, exactly the reverse takes place. The patient looks, not to the side of the lesion, but away from it, or towards his spasmodically contracted limbs, if, as often happens, a Jacksonian attack occurs simultaneously. Frequently the head is turned spasmodically in the same direction as the eyes (*vide* Fig. 81).

As a conjugate deviation towards the side of the lesion is also observed after destruction of the angular gyrus, it might be thought that there was here a second ocular centre. This is, however, not the case. The angular gyrus is, rather, merely a thoroughfare for fibres which connect the visual area in the cortex with the frontal centre for the ocular muscles. No less important, from the point of view of regional diagnosis, is the fact that conjugate deviation may also occur as a result of lesions of the parietal lobe.

Irritative phenomena in the sensory sphere also accompany affections of the senso-motor cortical area. Paræsthesias have not infrequently been observed

in the monoplegic muscular area. Jacksonian attacks are very frequently ushered in by such paræsthesias (*sensory aura*); more rarely a lightning-like pain darts through the same area that is afterwards affected by convulsions. Broadly speaking, however, no very acute sensitiveness to pain is to be attributed to the cerebral cortex. The fearful headache from which so many patients with brain disease suffer is not an irritative symptom to be referred to the cortex, but is rather to be ascribed to the extremely sensitive meninges. If the state of tension in the latter, due to increased intracranial pressure, be diminished by trephining or lumbar puncture, the pains also abate. Exacerbation of the pain on percussion of the skull over the central convolutions, etc., is, however, sometimes observed in tumours of the senso-motor area.

(*b*) Motor and Sensory Disturbances of Subcortical Origin.

A result of the fan-shaped or conical arrangement of the motor and sensory fibres in the corona radiata (rapid convergence of pyramid fibres and divergence of thalamo-cortical fibres) is that the deeper the situation of a lesion between the cortex on one side and the internal capsule and basal ganglia on the other, the more nerve tracts will be destroyed by it in proportion to its size.

The crossed motor disturbances, therefore, due to lesions in the corona radiata are, contrary to those produced by cortical and capsular lesions, of more than monoplegic, if not of definitely hemiplegic, type. As, however, lesions in the corona frequently interrupt the important commissural tracts of the corpus callosum, their symptom complex sometimes includes the condition known as *apraxia*, which is characteristic of lesions of that body (*vide infra*, Chapter V.).

As regards sensory disturbances, their intensity and extent is generally proportional to the depth below the cerebral cortex at which the lesion producing them is situated. A preponderance in the hands and feet is generally to be observed either *ab initio* or coincidently with an abatement of the symptoms of sensory disturbance.

"Capsular" paralyses may next be considered. Among other causes these are present in the great majority of cases of cerebral hæmorrhage due to rupture of the arteria lenticulo-striata, of which special mention was made on p. 157. Fig. 75 shows that in the posterior limb of the internal capsule all the motor tracts for the opposite side of the body are closely packed together into a bundle of relatively small calibre. Total hemiplegia is, therefore, in by far the larger number of cases, the result of lesions of this posterior limb of the capsule. We have, that is to say, crossed paralysis of the lower facial, the hypoglossal, the upper and the lower extremity. If the lesion extends to the posterior third of the posterior limb, where the sensory tracts, immediately after their emergence from the optic thalamus, lie in a compact bundle, we have, in addition, *complete crossed hemianæsthesia.*

Occasionally, however, the lesion is limited to the posterior extremity of the internal capsule, the *carrefour sensitif*. The simultaneous interruption, in such a case, of the sensory, auditory, and visual tracts leads to the development of the triad symptom complex—*hemianæsthesia, hemianakusis*, and *hemianopia*. It has been stated that hemianosmia and hemiageusia (γευω, I taste) may also be present in these cases, but no sufficient proof of the statement has been given. Spastic paresis of the anæsthetic lower extremity is not uncommon, as the pyramid fibres therefor are in close contiguity with the anterior portion of the sensory neuron complex in the posterior third of the capsule.

The internal capsule is of such small dimensions that the development of monoplegias is almost impossible, and they are of extreme rarity in these cases.

We may now state briefly the *most important distinguishing characteristics of cerebral hemiplegia*.

1. As has been stated several times, the bilaterally innervated muscles (mastication, deglutition, larynx, eyes, trunk, and upper facial) escape. With regard to the upper facial, however, a slight impairment of contractile power is often noticed in the frontalis and orbicularis palpebrarum of the side opposite to the lesion. The contralateral eyebrow shows a tendency to droop, or the eye cannot be kept closed so long as is the case with the homolateral.

2. Even in fully developed hemiplegias, different groups of muscles are, as a rule, unequally affected. While, in a large number of muscles, power of movement gradually returns, others show, commonly, no sign of recovery (*peronei, flexors of knee, extensors of elbow, extensors of hand and fingers, external rotators of arm; supinators of forearm*). The arm is, as a rule, much more severely affected than the leg. In children the hand shows a much stronger tendency to recovery than in adults.

3. The positions adopted by the extremities in consequence of the preponderating action of the muscles which recover their motility are often, in later stages, fixed by contracture. As a result, the foot is held in the position of equino-varus, and the knee extended, so that, in walking, the leg must be dragged forward, the foot describing a lateral semicircle (*circumduction, helicopody*). The arm is fixed in adduction, the elbow in flexion, the forearm in pronation, and the hand and fingers in flexion (*vide* Fig. 82). These contractures are thus produced: the ventral horn cells, freed from the inhibiting influence of the pyramids, are tonically stimulated by the impulses flowing in constantly by way of the posterior roots, and a summation of these stimuli occurs. The striking fact that it is certain definite muscles which recover their motility, and are thus (thanks to the supranuclear, hypertonic character of cerebral hemiplegia) predisposed to contracture, depends on some conditions of innervation, the anatomy of which is not yet fully known to us. Probably the subcortico-spinal tracts (pp. 4 and 5) from the *thalamus*, the *tegmentum*, the *nucleus of Deiters*, and the *roof of the mid-brain* are in particularly intimate relation

with the ventral horn cells of these groups of muscles, so that the reparatory efforts of the organism bring them again, with comparative ease, under the influence of the cerebral innervation, though by a circuitous route. Cases of cerebral hemiplegia in which muscular groups other than those mentioned gain the preponderance, and enter into contracture, so that, for example, the leg is fixed in flexion, form quite transitory exceptions.

FIG. 82.—LEFT HEMIPLEGIA DUE TO A HÆMORRHAGE INTO THE RIGHT CAPSULE (TYPICAL CONTRACTURES, CIRCUMDUCTION).

4. The hypertonus of cerebral hemiplegia is accompanied (unless there are complicating conditions which interrupt the reflex arc; *cf.* Cerebellar Tumours, p. 139) by exaggeration of tendon reflexes. To this hyperreflex, and to the other pathological reflex phenomena (*Babinski's, Mendel-Bechterew's, Oppenheim's and Bing's signs, cloni, associated movements,* etc.), the statements made in the section on the Spinal Cord apply (pp. 23-26). As an example of an associated movement characteristic of cerebral hemiplegia may be mentioned the so-called *platysma phenomenon,* an energetic, easily perceived contraction of the platysma on the sound side when the patient opens his mouth. The cutaneous reflexes, especially the *abdominal* and the *cremasteric,* are, on the contrary, almost always diminished, or even abolished, on the paralyzed side; this symptom may be called to our aid, when we have to deal with a patient who is lying unconscious after an apoplectic stroke, to determine on which side the hemiplegic paralysis will be present when he wakes from his coma. The explanation of this phenomenon is by no means clear. In the section on Segmental Diagnosis of Spinal Lesions we have attempted an explanation with which we must, in the present state of our knowledge, content ourselves. The conjunctival reflex is also often absent on the paralyzed side.

5. The paralysis is not degenerative in type. There are, however, exceptional cases in which the so-called *cerebral atrophy* sets in in the paralyzed, or even in the recovered, muscles—an atrophy which cannot be referred to disuse, and which is accompanied by diminution of electrical excitability, if not

by the reaction of degeneration. *Arthropathies* have also been occasionally observed. Still more rarely atrophy of bones on the paralyzed side has been recorded. More frequent than any of these trophic disturbances are *sympathetic disturbances of vaso-motility and of the sweat function*, for the physio-pathological explanation of which we may refer to the section on the Spinal Cord (pp. 20-23).

6. In hemiplegias resulting from cerebral hæmorrhage, fairly complete and rapid recovery from the paralysis often takes place. This is the case when the hæmorrhage has not severed the cortico-spinal tracts, but has occurred in their neighbourhood—in the lenticulate nucleus, for instance. The condition of tension produced for a time in the neighbourhood of the hæmorrhagic focus, the collateral œdema, etc., are, in such cases, responsible for the indirect paralytic effects produced in the region of the motor tracts. Only those pareses which are still present six to eight months after the attack are to be regarded as direct focal symptoms, and to be estimated accordingly in prognosis.

7. Immediately after the "stroke," as a shock or "diaschisis" effect, the tendon reflexes are, as a rule, abolished, and the muscles become flaccid. When, in spite of the coma, the reflexes are exaggerated and the muscles spastic, we are justified, in view of recorded experiences, in giving with considerable confidence a diagnosis of *hæmorrhage into the lateral ventricle*—a very grave condition. No satisfactory explanation of this fact can at present be given.

Some *atypical forms of cerebral hemiplegia* deserve mention.

(*a*) The so-called *hemiplegia sine materia*. Cerebral hemiplegias which, apart from the typical case of hæmorrhage, may be caused by foci of softening, by tumours, by the formation of cavities, sclerotic conditions, etc., have also occurred occasionally without any lesion in the central nervous system being discoverable on post-mortem examination. Many of these cases have been those of nephritic patients who have died of uræmia. The uræmic poison seems here to have, for some reason at present quite obscure to us, exercised a paralyzing action on one side only, just as it may exercise a unilateral irritating action and cause Jacksonian convulsions. The earlier authors imagined an arterial spasm of one side, and spoke of *apoplexia serosa*. Other cases of so-called hemiplegia sine materia, recorded in the older medical literature, depend probably on inadequate investigation, and are to be reckoned as examples of local softening; or, again, we may have to do with patients suffering from an undiagnosed dementia paralytica who have died in one of the "apoplectiform attacks" characteristic of this disease.

(*b*) The so-called *lacunar hemiplegia*. We have to do here, not with a massive lesion which interrupts the cortico-spinal tracts, but with disseminated, mostly miliary, lesions, small areas of softening, and capillary hæmorrhages, whose physio-pathological basis is cerebral arterio-sclerosis. This form of hemiplegia is marked by a great power of rapid recovery and very slight liability

to the development of contractures, but also, on the other hand, by a pronounced tendency to recurrence in the originally free hemisphere. In the latter case a spastic-paretic condition comes on in the bilaterally innervated muscles, and reveals itself by phenomena suggestive of pseudo-bulbar paralysis. Sensibility and cutaneous reflexes are, in this form, hardly affected. Even if the onset be sudden, the symptoms are not of severe type; consciousness is generally retained or only slightly clouded, and, if lost completely, the unconsciousness is of short duration, generally less than an hour.

(*c*) *Chronic progressive hemiplegia*, which develops quite gradually, either as the result of tumour growth in the internal capsule or of advancing closure of bloodvessels due to arterio-sclerosis. In the latter case it is distinguished from lacunar hemiplegia by its progressive course and absence of any tendency to recovery. Very rarely it may happen that chronic morbid processes, such as cortical atrophies (*e.g.*, in epileptics), give rise to the development of the clinical picture of this chronic progressive hemiplegia by interrupting the connection between the pyramid tracts (which themselves remain unaffected), and their related cortical cells and cell-groups. This condition is known as *intracortical hemiplegia*.

(*d*) The so-called *homolateral hemiplegia*. A considerable number of cases have been recorded in medical literature in which the lesion has been found, not on the opposite side to the paralyzed muscles, but on the same side. In most cases of the kind we have to do with faulty clinical or pathological observation. Thus, irritative phenomena may appear in the paralyzed extremities very soon after the stroke (*vide infra*), and be mistakenly regarded as evidences of voluntary action; while the opposite limbs lie flaccid and inert, in consequence of the coma, and are thought to be paralyzed. Further, in the post-mortem examination, the attention may be so drawn to a lesion of one hemisphere, which does not involve the motor tracts at all, that less obvious lesions, say in the pons or medulla of the opposite side, escape observation altogether. When, however, all allowances have been made for these cases erroneously described as cases of "homolateral hemiplegia," some cases remain which are above suspicion. In some instances of the kind, *absence of the pyramid decussation*, a comparatively rare anomaly, has been proved. Further, a tumour may push one hemisphere so far over to the opposite side that the substance of the other is very seriously compressed and injured, and rendered ischæmic in a high degree.

For the differential diagnosis between cerebral and peduncular, pontine and bulbar hemiplegias, which are, as a rule, distinguished by their *alternating* type, reference may be made to the account of alternating hemiplegias given on p. 95 *et seq.* The development of crossed hemiplegia, as the result of lesions at the level of the pyramid decussation, is explained on p. 92. Unilateral lesions which cut across the cortico-spinal tracts at points still

farther caudal reveal their spinal situation by the *symptom complex of Brown-Séquard.*

Irritative symptoms affecting the motor complexes of the internal capsule are, in the main, late symptoms. When an attack of apoplexy comes on, but especially at the moment when embolic plugging of a vessel occurs, spasms may come on in the muscles which are to be the seat of paralysis later on, as the result of irritation of the area involved by the embolus. They are not in any way pathognomonic, as they may occur in irritation of the motor tracts in the crura cerebri, the pons, the medulla, and the spinal cord.

All the more typical, therefore, are the late hyperkinetic phenomena which come on when a capsular hæmorrhage has affected the closely neighbouring basal ganglia; the outer zone or putamen of the lenticular nucleus, the caudate nucleus, and probably also the optic thalamus being especially in question (*vide infra*, Chapter V.). We allude to unilateral movements of the nature of *chorea* or *athetosis*, which can only in pure and typical cases be sharply differentiated from each other.*

Fig. 83.—Athetotic Movements of the Hand.

In *post-hemiplegic hemichorea*, the extremities are the subject of lively jerking and shaking movements ("*hemiballismus*"), which cease in sleep, but cannot be suppressed by voluntary effort—are, in fact, exaggerated by any attempt so to suppress them. In *hemiathetosis post-hemiplegica*, slow movements occur in the hands and feet, but especially in the fingers and toes. The movements begin with hyperextension, and recall the movements of the tentacles of a cuttle-fish. They do not always cease during sleep, but they can, to a certain extent, be suppressed by voluntary effort (*vide* Fig. 83). In many cases, however, the irritative symptoms noticed constitute a mixture of hemichorea and hemiathetosis, or, rather, a transition from the former to the latter. The latter is possibly merely a variety of the former, the more frequent variety; for true unmixed hemichorea, as above described, is a very rare condition.

Hemiathetosis and hemichorea usually commence some months after the onset of the hemiplegia, when some degree of voluntary movement has been

* It has been pointed out, when speaking of Benedikt's syndrome (*vide supra*, p. 95), that a lesion of the red nucleus of the tegmentum—closely connected with the putamen by the so-called *ansa lenticularis*—may excite choreic and athetotic movements. The choreic-athetotic phenomena present in lesions of the superior cerebellar peduncle (*vide supra*, p. 148) may also have relation to the anatomical and physiological interdependence of that structure and the red nucleus.

recovered. A very rare condition is "*hemichorea præhemiplegica*," which has been observed in some slow hæmorrhages into the optic thalamus.

Chorea-athetosis has occasionally been ascribed to irritation of the motor cortex (by hæmorrhage or meningitis); most probably, however, small foci in the basal ganglia or the mid-brain have been overlooked at the autopsy. Generally speaking, these hyperkineses are not to be regarded as true irritative symptoms, but as expressive of a withdrawal of normal inhibition.

Childhood is a period especially liable to the development of hemichorea and hemiathetosis. The latter symptom is found in 50 per cent. of the cases of infantile cerebral hemiplegia.

A rare post-hemiplegic irritative symptom is *post-hemiplegic tremor*, which resembles, sometimes the "intention" tremors of multiple sclerosis, sometimes the rhythmic oscillatory contractions of paralysis agitans. Whether it is always to be ascribed to an affection of the globus pallidus of the lenticulate nucleus, as has been maintained, is, in my opinion, questionable (*vide infra*, Chapter V.).

CHAPTER III.

LOCALIZING VALUE OF VISUAL DISTURBANCES.

1. The Optic Tract (*vide* Fig. 84).

From the retina, where they connect with the special sensory epithelium, the so-called "rods" and "cones," the optic fibres pass, in the optic nerve trunk, to the optic chiasma. Here a partial decussation takes place, of such a nature that, of the optic tracts leaving the chiasma, that on the right side contains the fibres from the right halves of the two retinæ, while that on the left contains the fibres from the two left halves.

The physiological result of this anatomical arrangement, combined with the fact that the lens projects on to the retina an inverted image of external objects, is that perception of the left half of the field of vision is conveyed by the right optic tract, and *vice versa*.

The fibres which cross at the chiasma come from the mesial (nasal) portions of the retinæ, and form the greater part of the whole body of optic fibres. The lateral halves of the fields of vision, whose impressions stimulate these decussating fibres, are somewhat larger than the mesial, which are restricted by the bridge of the nose.

Each optic tract carries the optic nerve fibres in a dorsal direction, winds round the cerebral peduncle, and passes to the primary visual centres of the same side, where the fibres terminate These centres are: the posterior portion of the optic thalamus (*pulvinar thalami*), the lateral geniculate body, and the superior quadrigeminal body.

From the primary visual centres the stimuli received from the retina are further conveyed in two directions:

1. The central visual tract, or bundle of Gratiolet, *tractus thalamo-occipitalis*, passes to the cortical visual area of the same side—*i.e.*, to the *cuneus* and the upper portion of the gyrus lingualis—on the mesial surface of the occipital lobe.

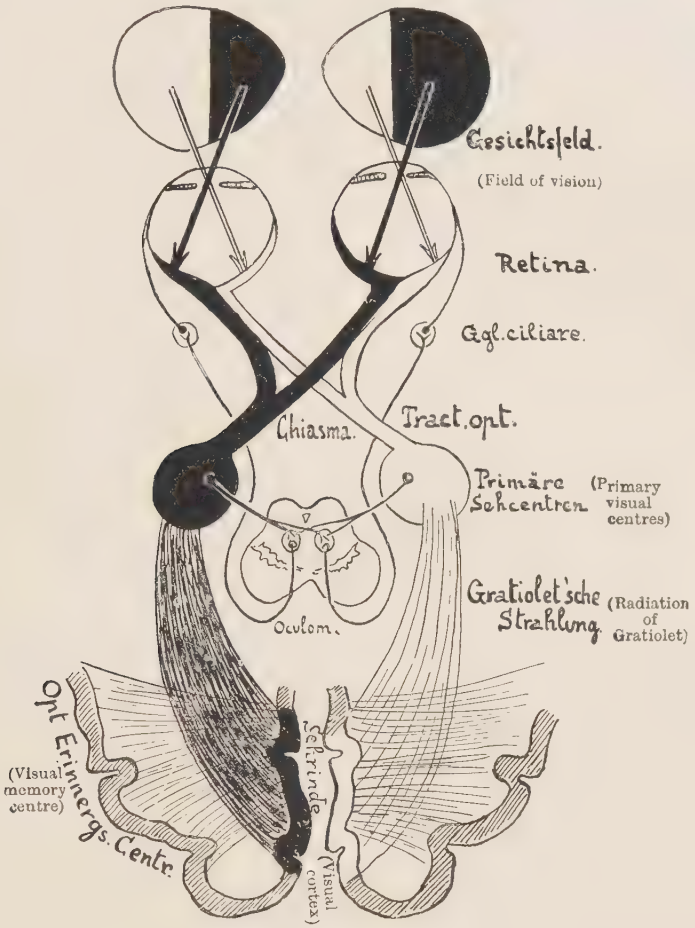


FIG. 84.—VISUAL TRACT AND PUPILLARY REFLEX TRACT.

The cortical visual centre of each hemisphere, therefore, known as the "area striata,"* receives perceptions from the *contralateral halves of the fields of vision.*

2. From the superior corpus quadrigeminum, nerve fibres pass to the small-celled lateral nucleus (*nucleus of Edinger and Westphal*) of the oculo-motor nerve

* Because of its histologically obvious striation due to Gennari's or Vicq d'Azyr's bundle. In certain races (Malays, Egyptians) the area striata extends to the lateral portion of the occipital lobe.

(*vide supra*, p. 127). It is by means of these fibres that reflex contraction of the pupil on stimulation of the retina by light is brought about. The pupillary reflex arc is built up of four sets of nerve fibres: (*a*) From the retina to the superior corpus quadrigeminum; (*b*) from the superior corpus quadrigeminum to the Edinger-Westphal nucleus; (*c*) from the latter to the ciliary ganglion; and (*d*) from the ciliary ganglion to the sphincter pupillæ. The connection (*b*) is both crossed and homolateral; that is to say, each corpus quadrigeminum sends fibres to both Edinger-Westphal nuclei. Illumination of a single eye, therefore, causes contraction, not only of its own pupil (*direct reaction*), but also of that of the other eye (*consensual reaction*). The nature of the lesion on which depends the form of sluggishness of the pupillary reflex found in tabes and paralysis, and known as the *Argyll-Robertson phenomenon*, is not yet fully established; most probably it has its seat in the connecting tracts between the primary visual centres and the Westphal-Edinger nucleus.

From the area striata, association fibres pass to the lateral convex surface of the occipital lobe and the inferior parietal lobe. As the area striata is the perception centre for optic impressions, so are the latter areas of the cortex centres for visual concepts and the storing up of memory pictures of visualized objects.

2. Localization of Interruptions of the Optic Tract.

A knowledge of the anatomical conditions governing the course and distribution of the optic fibres enables us in many cases to determine with considerable precision the situation of a focal lesion in the region of the optic conducting tracts (*cf.* Figs. 85-88).

A. Lesions of the Optic Nerve.—Total destruction of the optic fibres at a point peripheral to the chiasma (Fig. 85) produces, of course, complete blindness—amaurosis—of the one eye. Pupillary reflexes are not evoked by the affected eye, but may be by the other, whose perception of light is unimpaired. If only a portion of the optic fibres is destroyed, we have, instead of amaurosis, scotomata, narrowing of the field of vision, etc., pathological phenomena we need not consider more fully here, as they belong to the domain of ophthalmology.

B. Lesions of the Chiasma.—(*a*) If the lesion involves only the mesial portion of the chiasma (Fig. 83), as is the case in tumours of the hypophysis, hydrocephalus of the third ventricle, dilatation of the infundibulum, and empyema of the sphenoid antrum, it destroys the decussating fibres which come from the nasal portions of the retinæ. The result is to blot out both temporal halves of the field of vision, and to cause the condition known as *bitemporal heteronymous hemianopia* or *hemiopia*. *Heteronymous*, because the right eye is blinded for the right and the left eye for the left half of the field of vision.

(*b*) If, on the other hand, the external portions of the optic nerve decussation are destroyed, the mesial remaining unaffected (Fig. 86)—a very rare condition which may be brought about by aneurysm of both carotids, symmetrical gummatous foci at the base of the skull, perhaps also by cysts of the hypophysis bulging laterally, etc.—we have suppression of function in the outer portions of the fundi oculorum, and, as a result, *nasal heteronymous hemianopia.*

Fig. 85.—Production of Bitemporal Heteronymous Hemianopia by a Lesion of the Mesial Portion of the Chiasma.

(*c*) The result of destruction of one-half of the chiasma (Fig. 87) is blindness on the side of the lesion, and temporal hemianopia in the other eye.

C. Lesions of the Optic Tract, the Primary Visual Centres, and the Radiation of Gratiolet.—All these lesions cause *homonymous lateral hemianopia* for the halves of the field of vision contralateral to the lesion. *Homonymous,* because either both right or both left halves of the visual field are involved.

(*a*) If the lesion is peripheral to the point at which the reflex fibres to the Edinger-Westphal pupillary nucleus are given off (Fig. 88), a ray of light thrown

on to the blinded halves of the retinæ causes no contraction of the pupil (*hemianopic pupillary inactivity*); its access to the oculo-motor nucleus is blocked.

(*b*) If the lesion is central to the point at which the above reflex fibres are given off, illumination of the insensitive halves of the retinæ does, on the contrary, cause contraction of the pupil (*hemianopic pupillary reaction, Wernicke's phenomenon*), because the stimulus, while unable to reach the cortical visual centres, is not barred from the centres for the iris sphincters.

FIG. 86.—PRODUCTION OF NASAL HETERONYMOUS HEMIANOPIA BY A LATERAL LESION OF THE CHIASMA.

D. Lesions of the Area Striata (Cortex of the Cuneus and Upper Part of the Gyrus Lingualis).—A unilateral lesion (Fig. 88) has, of course, if the whole visual area of the cortex is destroyed, the same result as an interruption of the corresponding thalamo-occipital fibres—*homonymous lateral hemianopia* with *retained pupillary reaction*. If only a part of one visual area be destroyed, a part only of the contralateral halves of the visual fields is blotted out—the so-called *quadrant hemianopia*. If the affection is bilateral (Fig. 88)—

tumours of the falx cerebri, war wounds, toxic disturbances of function in the cortex, such as uræmia and saturnism—we have *cortical blindness*, in which the pupillary reaction remains unaffected, and can be evoked in both amaurotic eyes.

E. Lesions of the Convexity and of the White Matter of the Occipital Lobe and of the Gyrus Angularis.—Bilateral lesions involving the visual memory

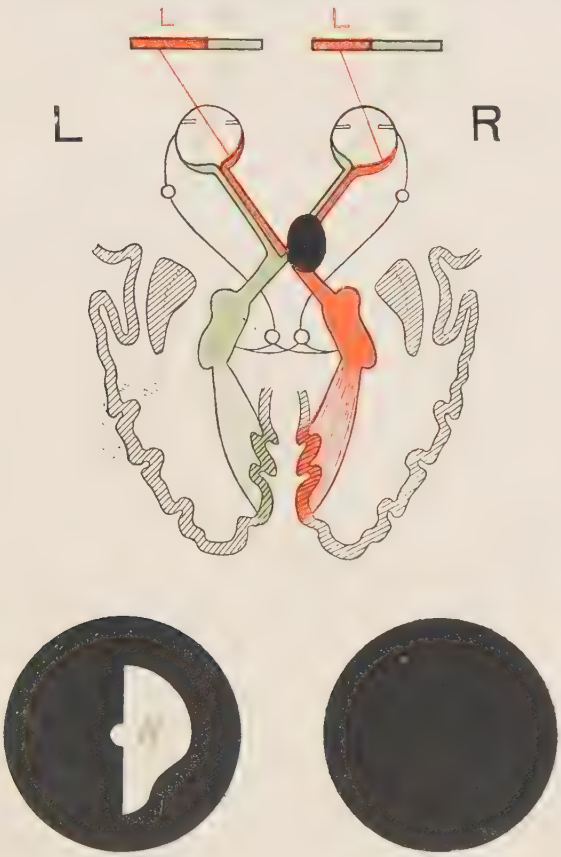


Fig. 87.—Production of Amaurosis of the Homolateral and Temporal Hemianopia of the Contralateral Eye by Destruction of One Half of the Chiasma.

centres in the cortex, or the association fibres which pass thither from the area striata (Fig. 89), cause *conceptual* blindness (" *mind-blindness*," " *object-blindness* ")—a condition in which patients can see objects as flat surfaces or as solid bodies, but are unable to recognize them for what they are, or to state their objects and uses. Where unilateral disease of the occipital lobe causes conceptual blindness, we have to do with tumours growing in one and pressing on the other occipital lobe. The left angular gyrus has a special function. This

is the centre for recognition of the meaning of written signs, of "*letter pictures*"* —a centre which has close relations, not only with the visual cortical area, but also with the cortical area for the understanding of speech (*vide* Figs. 73 and 95). Lesions of the angular gyrus (Fig. 89) cause, therefore, *alexia, word-blindness,* inability to read, sometimes also conjugate deviation (*vide supra,* p. 163).

If the cuneate cortex is destroyed in one hemisphere, and the occipital white matter or the cortex of the occipital convexity in the other, hemianopia

FIG. 88.—PRODUCTION OF LATERAL HOMONYMOUS HEMIANOPIA BY INTERRUPTION OF AN OPTIC TRACT.

is complicated by conceptual blindness; for the unaffected area for visual memory is put out of action, owing to the fact that no stimuli reach it from the neighbouring cuneus.

It should be added that, in the regional diagnosis of visual disturbances, any accompanying symptoms of a different order must be considered. If,

* This expression, though convenient, is naturally to be taken *cum grano salis,* since the existence of a "centre" for an acquired faculty such as reading can hardly be admitted in the same sense in which one speaks of centres for inborn functions such as flexion of a joint, etc.

for example, an homonymous lateral hemianopia comes on in the course of a meningitis, we may infer that the contralateral cuneate cortex is involved; if, in a case of cerebral softening, we have alexia with right-sided homonymous hemianopia and Wernicke's phenomenon, the inference is that a focus of softening in the angular gyrus has, as not infrequently happens, spread to the subjacent fibres of Gratiolet (Fig. 89); if, on the other hand, hemianæsthesia and hemianakusis are present, the focus is to be looked for in the *carrefour sensitif* (*vide supra*, p. 165).

The foregoing conditions, however, by no means exhaust the list of regional diagnostic criteria at our disposal in dealing with disturbances of vision. We would draw attention in the first place to the remarkable clinical differentia

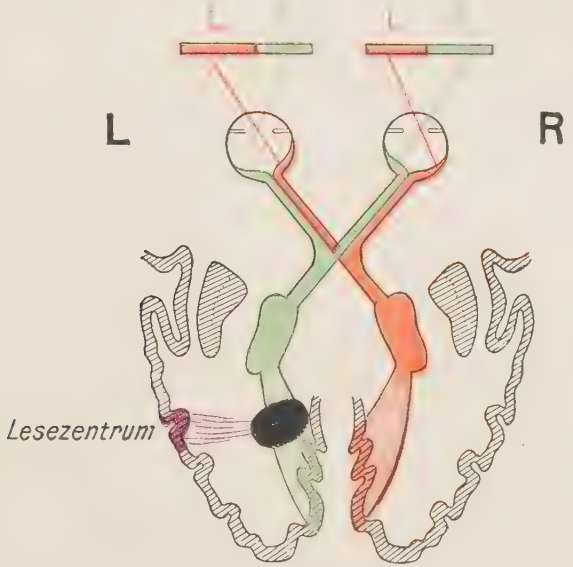


FIG. 89.—PRODUCTION OF RIGHT-SIDED HOMONYMOUS HEMIANOPIA WITH ALEXIA BY A LESION IN THE WHITE MATTER OF THE LEFT OCCIPITAL LOBE.

between the homonymous hemianopias due to lesions of the optic tract, and those due to affections of the occipital lobes—namely, that in the first case the obliterated part of the field of vision usually makes itself obvious as a positive skotoma,* while in the second case the skotoma is always negative. Thus in lesions of the tract the patient is usually aware of a black area or patch covering one-half of his field of vision, while in cortical or subcortical disturbances of the visual apparatus in an occipital lobe he is no more spontaneously conscious of the defect in his field than is the normal individual of his Marriott's "blind

* Unfortunately this distinguishing feature, though a frequent, is not a constant or inevitable criterion of tract hemianopias, but is largely dependent on the powers of observation and degree of education of the patient. The defect in the visual field is probably not subjectively perceived in cases of disease of the lateral geniculate body.

spot." Such occipital hemianopias first reveal themselves on examination with the perimeter, unless, clinically, some peculiarity in the patient's behaviour has enabled us already to detect them. Such patients, for example, often keep the head turned instinctively towards the side of the obliterated field, that as

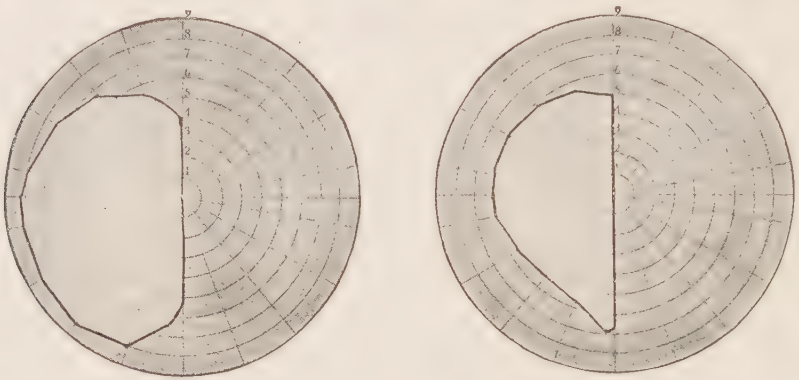


FIG. 90.—LESION OF THE LEFT OPTIC TRACT : COMPLETE RIGHT HOMONYMOUS HEMIANOPIA (FRACTURE OF BASE).

much as possible of the picture of their surroundings may fall on the percipient half of the retina, and occasionally, in consequence, feel as if they had a strabismus towards the side of the obliteration. If a sheet of white paper with a black line on it is put before such a patient, and he is asked to divide the line in half, he will divide, not the whole line, but only that part lying in his reduced field of vision. If, within the anopic half of the field, a finger is

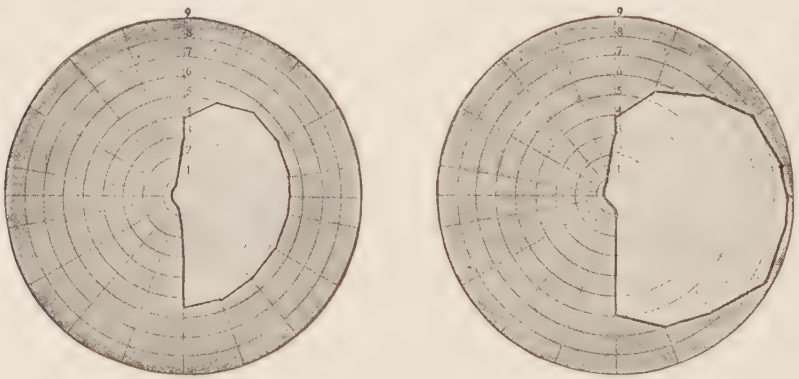


FIG. 91.—LESION OF THE RIGHT OPTIC TRACT : LEFT HOMONYMOUS HEMIANOPIA.

suddenly advanced towards his eye, there is no blinking of the eye; while, if the other side be experimented on, there is an immediate blink as a protective reflex.

This difference between the tract hemianopic and the cerebral hemianopic

in the subjective recognition of the damage suffered has its objective correlative in the results of perimeter tests. A tract hemianopia may be complete, and the dividing line between intact and obliterated halves of the field of vision pass perpendicularly through the fixation point; usually, it is true, the line of

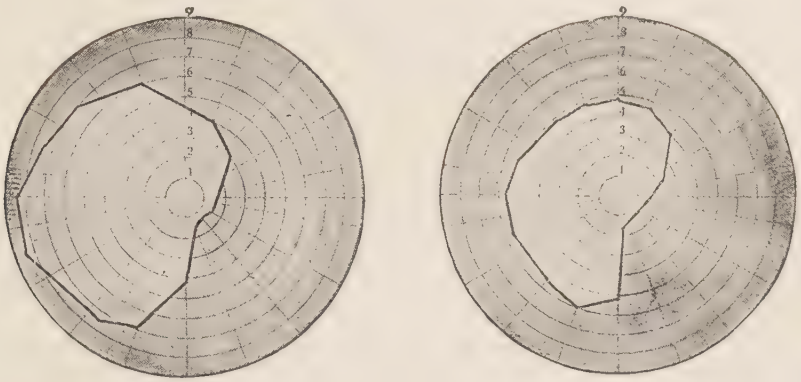


FIG. 92.—RIGHT HEMIANOPIA WITH OVERLAPPING FIELD OF VISION OF WHICH THE PATIENT IS UNCONSCIOUS: DESTRUCTION OF OCCIPITAL WHITE MATTER BY PORENCEPHALO-HYDROCEPHALUS (TRAUMATIC) OF LEFT HEMISPHERE.

demarcation winds round the fixation point, so that "central sight" remains intact. In subcortical and especially in cortical lesions of the occipital lobe we have, on the contrary, many incomplete hemianopias in which, on the far side of the middle line, a more or less extensive zone of retained vision juts out

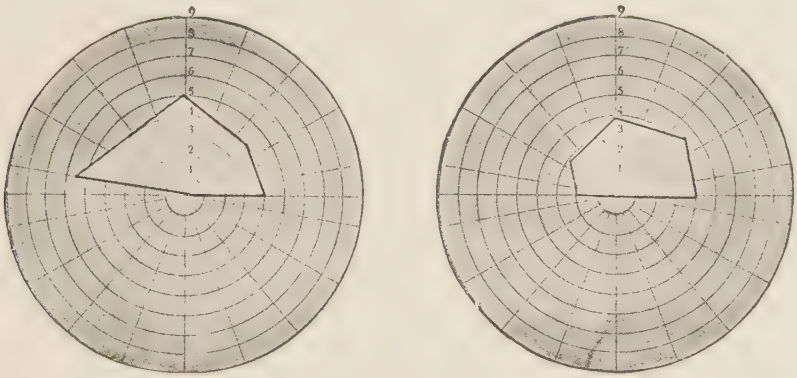


FIG. 93.—HEMIANOPIA HORIZONTALIS INFERIOR: (TRANSVERSE GUNSHOT WOUND OF OCCIPUT).

like a peninsula or cape into the obliterated area; this symptom, of extreme importance in regional diagnosis, is known as the "overlapping" field of vision (*vide* Figs. 90-92).

The explanation of the persistence of central sight in the great majority of cases of hemianopia, as also of the "overlapping" field, would appear to lie

in the fact that the macula and its immediate neighbourhood have in many individuals not an insular, but a somewhat diffuse, and in many human subjects a bilateral representation in the *area striata*. Occasionally, however, on the other hand, homonymous central scotomata come under observation, which are due to small circumscribed injuries of a cuneus (*e.g.*, from small splinters of bone driven into the brain). In such patients there can be no question of a bilateral or even of a diffuse cortical representation of the macula.

Quite characteristic of the clinical histories of patients with suddenly developed unilateral lesions of the cuneus (hæmorrhages and emboli) is the

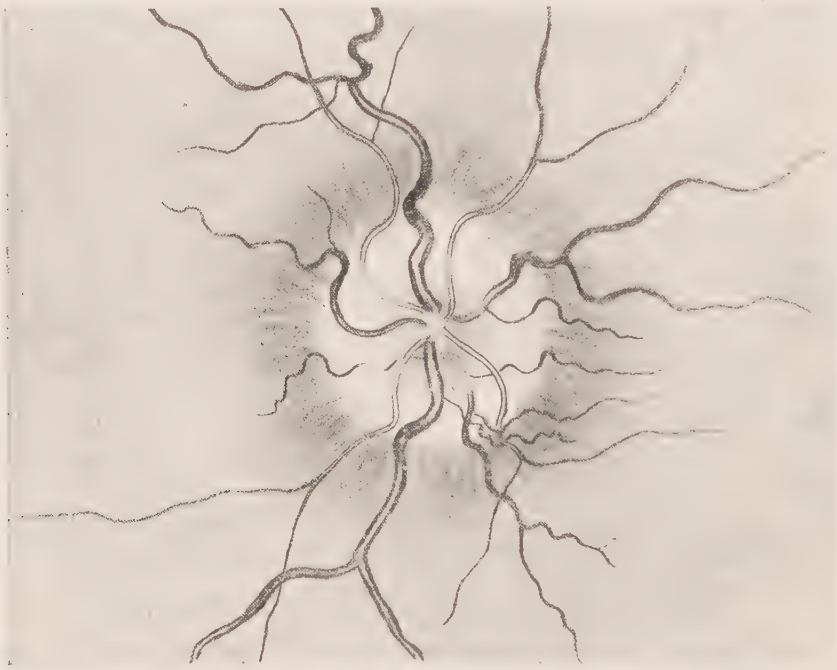


Fig. 94.—Choked Disc (Sarcoma of Left Cerebellar Hemisphere).

statement that the attack caused at first complete blindness in both eyes, this being replaced secondarily by homonymous hemianopia. It would seem that a transitory inhibition of function in the intact visual centre as the result of shock or "diaschisis" is the condition underlying this transitory initial symptom which is never seen in tract hemianopias.

As regards the regional diagnostic significance of the so-called "quadrant" hemianopia (*vide* p. 174), it has been maintained that the upper lip of the calcarian fissure corresponds to the upper, the lower to the lower half of the retina, so that lesions of the dorsal lip of the calcarian fissure of one hemisphere cause a crossed hemianopia in lower quadrants (hemianopia horizontalis

inferior), while lesions of the ventral lip cause hemianopia in upper quadrants (hemianopia horizontalis superior). This statment of a rule is, however, only to be taken with qualifications. All that can be stated with certainty is that no case of simple lesion of the dorsal cuneus region is known in which the skotoma has involved only upper quadrants of the field of vision, and conversely no case of lesion exclusively of the ventral half of the cortical visual centre with only lower quadrants affected. The condition which may be found in a transverse gunshot wound through the upper part of the cuneus in both hemispheres is shown in Fig. 93.

Direct observation of the optic nerves with the ophthalmoscope is of very little assistance in localization. All the more importance, therefore, is to be attached to the condition known as "choked disc" (*vide* Fig. 94) as a general symptom pointing to increase of intracranial pressure. With headache, cerebral vomiting, vertigo, slowing of the pulse, and disturbances of consciousness, it plays a leading rôle in the symptom complex of pressure on the brain.

CHAPTER IV.

LOCALIZING VALUE OF DISORDERS OF SPEECH.

Disorders of speech due to focal diseases or lesions of the cerebrum must be distinguished as *anarthric* or *aphasic*.

The anarthric form, disturbance of articulation, depends on lesions of the nervous mechanism governing the muscles of speech. We meet it, therefore, in bulbar paralysis as the result of nuclear, in pseudo-bulbar paralysis as the result of supranuclear, lesions. The latter must be bilateral, as all the muscles in question—so far, at any rate, as their special speech function is concerned—are innervated from both hemispheres. At what point, however, in the cortico-nuclear nerve channels the bilateral interruptions may be situated, whether in the operculum, the centrum semiovale, the internal capsule, the cerebral peduncle, or the pons, is, so far as the development of anarthric or dysarthric disturbances of speech is concerned, a matter of indifference. A description of this form of disorder of speech has been given on pp. 108 and 109.

Very different is the case with the aphasias, which we classify as *motor*, or expressive, and *sensory*, or perceptive.

(*a*) In *motor aphasia* there is inability to translate ideas or concepts into words. The patient is not dumb; there is no paralysis of his muscles of speech. Their ordered co-operation, however, which is essential for normal speech, has become impossible for him; as in childhood he had to learn gradually how to use the organs of speech to form words, so now the kinæsthetic memory pictures for the concepts mastered during the process of learning speech have

become lost to him. The distinction between this form of disturbance of speech and the anarthric is expressed most briefly and significantly in the French language: "L'aphasique ne sait plus parler, l'anarthrique ne peut plus parler."

(*b*) In *sensory aphasia*, not the power to speak, but the understanding of speech, is lost. The patient hears the spoken word perfectly, but can no longer understand it, as it no longer evokes in the consciousness its corresponding concepts. His mother-tongue sounds to him as a foreign language, of which he has learnt little or nothing, sounds to a normal individual. He has lost his memory for the meaning of words. Sensory aphasia is often accompanied by *paraphasia*; that is to say, the patient makes continual mistakes in speaking, and uses, instead of the correct words, others—perhaps of similar sound. This

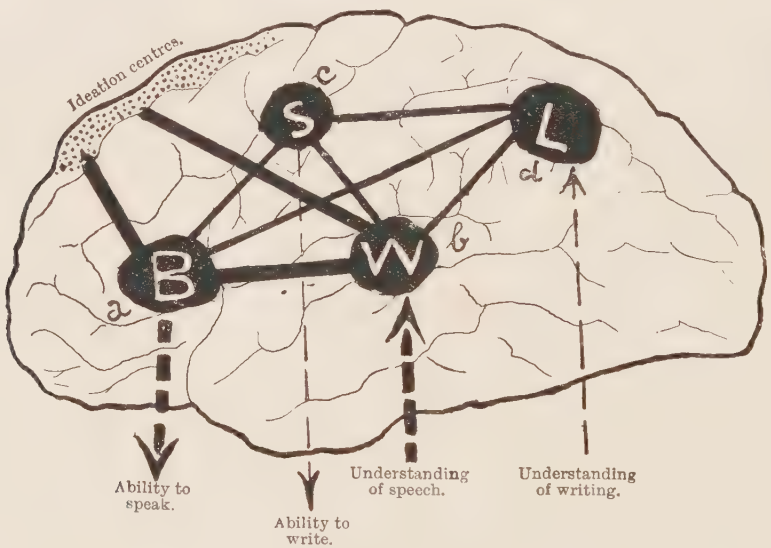


FIG. 95.—THE CORTICAL SPEECH CENTRES AND THEIR CONNECTIONS.

B = Broca's centre; W = Wernicke's centre; L = Reading centre; S = Writing centre; *a* = Inferior frontal convolution; *b* = Superior temporal convolution; *c* = Anterior central convolution; *d* = Angular gyrus.

is explained by the fact that unconsciously we test, so to speak, internally the "ring" of each word before uttering it by means of our sensory speech centre. If this no longer functions normally, the patient very readily uses incorrect words, and is unable to recognize his mistake, as, in consequence of his sensory aphasia, he does not understand his own words. Rarer is the combination of sensory with the so-called conceptual deafness or *acoustic agnosis*. The conceptually deaf person is not able to recognize a watch from its tick, a dog from its bark, a bunch of keys from its rattle, or a waterfall from its murmur. Besides the spoken word, in fact, other sounds of all kinds are no longer understood by him.

The motor speech centre (*anterior centre, Broca's centre*), which as the storing-up place for kinæsthetic memory pictures governs the co-ordination of the mechanism of articulation, is situated at the foot—*i.e.*, in the posterior portion—of the inferior frontal convolution, where it borders the operculum (*cf.* Fig. 95), but extends probably also into the neighbouring portions of the island of Reil, the second frontal convolution, and the pre-central gyrus. The *sensory speech centre* (*posterior centre, Wernicke's centre*), in which the tone pictures which render possible the understanding of speech are stored up, is situated in the left superior temporal convolution (Fig. 95, W).

If the anterior speech centre is destroyed by some morbid process, motor aphasia develops, while destruction of the posterior centre causes sensory aphasia. Should both centres be destroyed, both the ability to speak and the understanding of speech are lost, and we have the condition known as *total aphasia*.

A priori it would be natural to suppose that destruction of the nerve tracts coming from Broca's centre, or of those passing to Wernicke's centre, would have the same results as destruction of those centres themselves. It is not so, however, and it is possible to distinguish between cortical motor and sensory aphasia on the one hand, and subcortical forms on the other.

A detailed account of the symptomatology of these separate forms of aphasia would be out of place in an introduction to the study of regional diagnosis. It will suffice to establish the more important points:

As will be seen from Fig. 95, not only is the motor speech centre (B) connected with the cortical centres for the labial, lingual, and laryngeal muscles, and the sensory speech centre (W) with the auditory area in the cortex, but these two centres communicate also by means of association fibres: (1) with one another; (2) with the higher psychical centres of the frontal cortex, the so-called "ideation centres"; and (3) with the cortical centres for writing and reading (S and L). The gyrus angularis, the memory centre for the recognition of written signs, whose destruction causes alexia or word-blindness, constitutes a special reading centre (*vide supra*, pp. 175 and 176). A writing centre was formerly placed at the foot of the left middle frontal convolution; this localization is now recognized as erroneous, and the writing centre is regarded as identical with the centre for the hand and fingers in the precentral gyrus (Fig. 73, *c* and *d*).

This network of association fibres is of significance in connection with the psychical functions which we may briefly denominate "inner speech." By this term we understand all that must take place subconsciously in our brains before we put a thought into words and express these words in speech or writing, or before we gain full knowledge and understanding of the spoken or written expressions of others. This "inner speech" develops ontogenetically as follows: The child first repeats words he hears, then attaches concepts to them; then, when those concepts arise in his consciousness, he reproduces the words

himself; later, in his reading and writing lessons, connects sounds with the symbols chosen to express them, and so on.

Of primary importance for a true understanding of the question of aphasia is the fact that all the association fibres concerned in the function of "inner speech" run in the cerebral cortex, while those concerned in the act of speech itself pass through the medullary substance. This is shown diagrammatically in Fig. 96, from which it will be seen that a cortical lesion of Broca's centre (A) must rise give to more symptoms of disturbance of the speech function than a subcortical lesion (B). For by the former the association fibres for inner

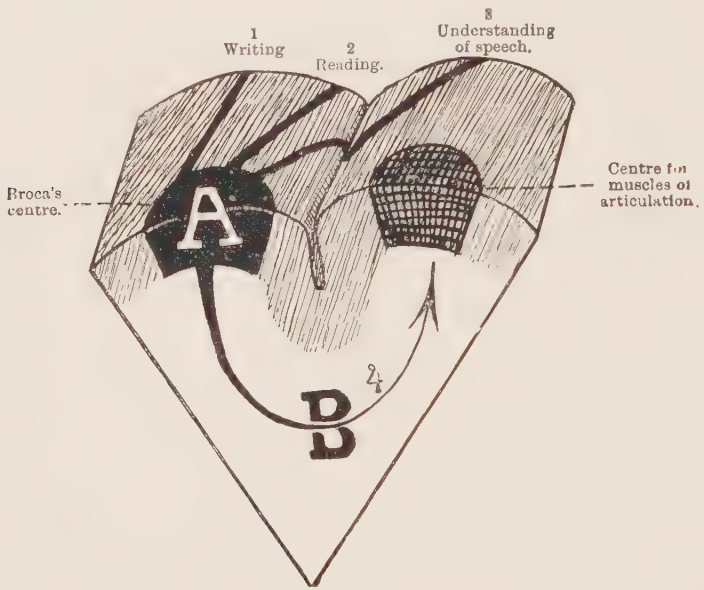


FIG. 96.

In a cortical motor aphasia (A), 1, 2, 3, and 4 are interrupted; in a subcortical motor aphasia (B), only 4 is interrupted.

speech are interrupted, while the latter in no way involves them. Thus subcortical motor aphasia is a pure aphasia, a simple inhibition of actual speech, a mere word-dumbness. *Mutatis mutandis,* cortical sensory aphasia is distinguished from subcortical, pure word-deafness, in a similar way. It may be laid down, therefore, as a general principle that *cortical aphasias are never pure.*

The sum of faculties we have denominated "inner speech" is impaired in highest degree in total aphasia. Here the patient has also lost the power either to use or to understand the language of gesture and facial expression (motor and sensory *amimia*).

The condition in which the individual has lost all power of mutual understanding with his fellows is known as *Asymbolia* or *Asemia.*

The agraphia and alexia which accompany cortical sensory aphasia are both very obvious. In cortical motor aphasia, on the other hand, only the agraphia is as a rule at all severe; the power of reading is much less impaired, and in many cases special methods are required for the detection of disturbances of the function. Printed words, for instance, may be understood, but not written words, or the patient may be unable to understand writing if the syllables are separated from each other, and so on. The disturbance of speech which characterizes cortical sensory aphasia is always obvious, and is of the nature of paraphasia; in cortical motor aphasia, on the other hand, the patient is, as a rule, not noticed to have any difficulty in understanding speech unless his interlocutor speaks very quickly or uses complicated sentences.

The case is different with aphasias due to subcortical affections of the left frontal or temporal lobe, which do not involve the cortex itself. In the former case (*frontal*), we have pure motor aphasia (=*word-dumbness*); in the latter pure sensory aphasia (=*word-deafness*). In neither case is there any defect in reading or writing; "inner speech" is intact; only the power to speak or the power to understand speech is lost. In pure word-deafness, too, there is no paraphasia, because the sensory cortical centre is able to exert its usual stimulating and controlling influence over the motor. In pure word-dumbness the patient, although he is unable to give utterance to words, has, in contrast to the patient with cortical motor aphasia, retained the corresponding kinæsthetic memory pictures, and is able to indicate with his fingers the number of syllables in a sentence to which he in vain endeavours to give utterance (*Dejerine-Lichtheim* phenomenon).

With a view to obviating mistakes in localization, attention must here be drawn to two further points: (1) Lesions situated very superficially in the subcortical region—*e.g.*, in the white matter of an individual convolution—may, in their effect, resemble cortical lesions, and give rise to "mixed" aphasias. (2) Probably, also, incomplete disturbances of the motor or sensory speech centres may exceptionally cause pure word-dumbness or word-deafness. In such cases we may assume that the association fibres between Broca's centre and the motor centre for the muscles of speech, or between the cortical area for the cochlearis and Wernicke's centre are interrupted intracortically, without destruction of function in the affected speech centres.

The further study of aphasia in all its minutiæ has, at present, little relevance to the subject of regional diagnosis. Special forms, such as transcortical aphasias, visual, tactile, amnesic aphasias, etc., which are developed when Broca's and Wernicke's centres are cut off from each other, from the cortical centres for the higher psychical functions (*ideation centres*), or from the sensory areas, cannot be ascribed to lesions of ordinary type, but must be regarded as depending on many varied and variable interruptions of nerve tracts. The

transcortical aphasias are, further, probably for the most part intermediate stages of cortical aphasias progressing towards recovery.

The so-called "optical aphasia" is observed in lesions of the white matter of the occipital lobe; we may conclude that the condition is due to an interruption of the connections between the centres for *recognition* of objects in the occipital lobe, and those for the *naming* of objects in the temporal lobe (Wernicke's tone-picture centre). One must be very much on one's guard against confusing this condition with conceptual blindness, and must satisfy oneself whether the patient really recognizes the objects he is unable to name. Such patients do not usually state that they are unable to name the object shown them, but name it incorrectly, the choice of a name being determined sometimes by some physical resemblance, sometimes again by a likeness in sound. Thus a candle may be called a pencil, scissors a pair of spectacles, or, on the other hand, grass may be called brass, and so on. As is usual with paraphasias, such patients are often unaware that they have used a wrong word, and only realize this when given an opportunity of estimating the object shown them by means of another than the visual sense—*e.g.*, a spoon by touch, a clock by the ear, sugar by taste, and so on.

The localization of the psychic speech mechanism in the left cortex is the rule only in right-handed individuals. Left-handed persons have their speech area in the right hemisphere. At the same time, cases have been recorded of motor aphasia in left-handed individuals as the result of lesions of the left inferior frontal convolution. It is no matter for surprise, therefore, and no argument against Broca's teaching, if right-handed individuals occasionally fail to become aphasic after destruction of the left inferior frontal convolution; they must be regarded as exceptional individuals, whose speech centres are on the right side. (It is, indeed, extremely probable that these cortical centres were originally bilateral.)

From the point of view of localization, we may sum up what has been said above concerning the study of disorders of speech as follows:

1. *Motor aphasia* points to a lesion of the *posterior inferior portion of the frontal lobe* on the left side in right-handed individuals, and *vice versa*.

2. *Sensory aphasia* points to a lesion of the *posterior superior portion of the temporal lobe* on the left side in right-handed individuals, and *vice versa*.

3. In the rare cases in which these aphasias are "pure"—*i.e.*, uncomplicated by defects of "inner speech," of reading and writing—a subcortical lesion, not very superficially situated, may be diagnosed with a high degree of probability. Exceptionally we may have to do with a minimal cortical lesion.

4. The rule of a left-handed localization in right-handed individuals, and *vice versa*, is subject to rare exceptions.

CHAPTER V.

I. LOCALIZING VALUE OF SOME RARER CEREBRAL SYMPTOMS.

(*a*) Auditory and Olfactory Defects due to Cerebral Lesions.

UNILATERAL destruction of the auditory region in the cortex (*vide* Fig. 73), or of the fibres passing to it from the inferior corpus quadrigeminum and the mesial corpus geniculatum, causes impairment of hearing or complete deafness on the opposite side. This crossed hemianakusis, which we have already alluded to as a symptom of lesions in the *carrefour sensitif*, is but transitory in character, a fact which forces us to the conclusion that each cochlear nerve is in connection with both auditory areas in the temporal cortex, though its relations with the contralateral one are the more intimate. The conditions are thus rather more complicated than would appear from Fig. 59. Permanent cortical deafness is caused only by complete destruction of the auditory area on both sides.

As regards the olfactory cortical area in the uncinate gyrus, it is certain that unilateral destruction thereof causes no olfactory defect, and that anosmia is produced only by complete destruction of this area on both sides. Of gustatory defects as symptoms of cerebral lesions nothing is known.

(*b*) Apraxia.

By apraxia is meant a condition in which the extremities, especially the hands, without being paralyzed, are unable to carry out certain purposive movements. The patient has lost his memory for the *synergias* and combinations of single movements necessary for the effective and suitable use of an instrument, for gesture, for greeting an acquaintance, etc. This condition has many analogies with motor aphasia.

Clinically, three distinct varieties of apraxia are recognized:

1. Ideational apraxia, a condition which resembles extreme absent-mindedness, and, as a general rule, only reveals itself in the performance of complicated movements. The power of forming in the ideational sphere a correct plan of action suited to the movements to be carried out is interfered with; the voluntary impulses are incorrect. The patient, for example, puts a match in his mouth with his cigar instead of lighting it, holds a seal in a flame and then presses it on the stick of sealing-wax, tries to cut the end of a cigar by nipping it between a match-box and its lid. The single movements necessary for these actions are for their part carried out quite correctly. The underlying anatomical condition is some diffuse morbid process, such as senile dementia, progressive paralysis, or diffuse cerebral arterio-sclerosis, so that this variety of apraxia has no regional diagnostic significance.

2. Motor apraxia. In this form the movements of a limb are carried out as if the patient were trying them for the first time; coarse movements are very clumsily executed, while finer manipulations (writing, threading a needle, sewing, etc.) cannot be executed at all. The underlying condition here is a lesion of the motor area governing the affected limb, a lesion which, while not leading to paralysis of the limb, effaces the kinæsthetic memory pictures on which its ordered movements depend.

3. Ideo-motor apraxia. In this form ideation and the motor centres for movements of the limb with their kinæsthetic memory pictures are both intact.

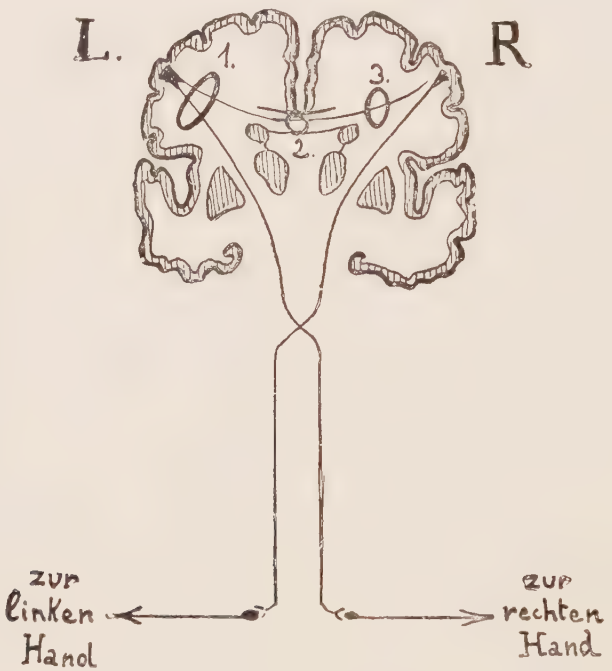


Fig. 97.—Three Foci which may lead to Apraxia of the Left Hand.
Focus 1 causes paralysis of the right hand also.

Their connection, however, with each other is interrupted. Simple movements whose execution is, so to speak, *laid down* in memory pictures in the motor centre, are, therefore, executed correctly, but when forming part of a larger movement sequence they are brought in *mal apropos* in incorrect relation with other elements of the sequence. The "orders" of the ideation centres no longer reach in correct form the motor centre of the limb, which has become to a certain extent independent of general cerebral control.

Examples: in the main it is a question of a substitution of one movement for another; the patient beckons when told to threaten with his fist, touches his

ear instead of his nose. Or, on the other hand, the movement passes over to a different section of the musculature; instead of giving his hand, the patient stands bolt upright. Or, finally, he makes so-called "amorphous" movements not resembling any purposive movements whatever, waves his arms about, spreads his fingers, and so on. In severe cases the motor perplexity is such that almost all movement is unattempted.

Ideo-motor apraxia is found under two separate sets of conditions:

Firstly, in general, when the senso-motor cortex, while retaining its integrity, is cut off from other cortical areas, especially from the centres for the storing up of verbal and material concepts, in the temporal and occipital lobes. Thus, lesions in the parietal lobe occasionally cause apraxia of the opposite hand. The "*apraxic*" agraphia, which may be present as one of the symptoms of such a lesion, is to be distinguished pathogenetically from "*aphasic*" agraphia.

In the second place, apraxia of the left hand may be observed when its senso-motor area in the right cortex, while remaining itself intact, has lost its connection with the senso-motor area of the left hemisphere. The left hemisphere reveals, at any rate in right-handed individuals, its functional superiority in this respect also, that its senso-motor area exercises a controlling influence over the activity of that of its fellow. This control is exercised solely by means of the commissural fibres of the corpus callosum. Apraxia of the left hand is found, therefore, in lesions which sever the callosal fibres or their prolongations, whether the interruption be in the corpus callosum itself* or in one of the two hemispheres. Thus, a lesion of the left centrum semiovale may not only cause right hemiplegia through interruption of pyramid fibres, but, simultaneously, a left-sided apraxia, owing to destruction of commissural fibres and consequent removal of the right senso-motor area from the controlling influence of the left (*vide* Fig. 97).

In the diagnosis of apraxia it is, of course, essential to exclude: (1) word-deafness—*i.e.*, to make sure that the patient understands the directions given him; and (2) agnosia—*i.e.*, to ascertain whether he recognizes objects correctly. One must also naturally be on one's guard against mistaking ataxic, choreic, or athetotic movements for apraxia. Motor and ideo-motor apraxia are occasionally associated with cataleptic phenomena (*vide supra*, p. 148).

The question of apraxia is not yet thoroughly cleared up; the whole subject, indeed, is still in process of investigation.

* If the apraxia is associated with a bilateral hemiparesis without hyper-reflex or Babinski's sign, it is probable that the interruption is situated in the corpus callosum itself. In proportion to the more or less median situation of the lesion is the intensity of the paresis alike or different on the two sides; there may also be a combination of unilateral hemiparesis with contralateral motor irritative symptoms—*e.g.*, convulsions or hemichorea. Sensibility remains intact as a rule in callosal lesions, as also the function of the cranial nerves; only when the most anterior portion of the corpus callosum is affected does one find a facial paresis produced.

(*c*) Disturbances in the Spheres of Intellect and Character.

Psychic disturbances are observed chiefly in lesions of the anterior portions of the frontal lobe. The lesion must be bilateral, or, if unilateral, it must be capable of producing a sympathetic symmetrical affection of the opposite frontal lobe (pressure from a tumour, etc.). In such a condition, pronounced intellectual defect of a similar kind to that found in progressive paralysis (in which there is marked atrophy of the frontal brain) is apt to come on, and, in addition, especially in tumours, a tendency to make nonsensical jokes (*moria*), with loss of moral sense and pleasure in offensive behaviour. Unilateral affections of the frontal lobes may be quite without symptoms.

II. SYMPTOMS DUE TO LESIONS OF THE BASAL GANGLIA.

Such sensory or motor disturbances as have been observed in lesions of the corpus striatum (caudate or lenticulate nucleus) must be regarded as due to the action of the lesions on the neighbouring internal capsule, seeing that identical symptoms may be produced by lesions in the neighbourhood of the capsule, which are situated neither in the lenticulate nor the caudate nucleus. The fact that bilateral lesions of the corpora striata may give rise to symptoms of pseudo-bulbar paralysis is also to be explained by the hypothesis of a disturbing action exercised by the lesion on neighbouring cortico-nuclear fibres.

It has been proved that lesions of the phylogenetically oldest portion of the corpus striatum, the globus pallidus, cause a muscular rigidity which is distinguished by the term "extra-pyramidal." All pathological reflexes which we have noted as characteristic of affections of the cortico-spinal tract (*vide supra*, p. 24 *et seq.*) are to be distinguished from this type of muscular rigidity, the most frequent example of which is found in Parkinson's disease.

For an explanation of the mechanism of this rigidity reference may be made to Fig. 98, which shows in diagrammatic form how the globus pallidus, through the fibres of the so-called "ansa lenticularis" which it sends to the red nucleus of the tegmentum, exerts a tonus-inhibiting action on the cerebellar tonus mechanism mentioned above on p. 139 *et seq.* (Figs. 67 and 68). The afferent limb of this complicated reflex arc is formed by the medium length elements of the posterior roots and the spino-cerebellar tracts; in the efferent limb, however, is included, as a most important "halting-place," the red nucleus, which sends the rubro-spinal tract to the spinal motor nuclei. If this inhibitory influence is done away with, the result is that the cerebellar tonus gains the upper hand, and this gives an entirely different picture from that of the heightening of spinal reflex tonus which is brought about by cutting out the influence of the pyramid tracts.

Besides the above-mentioned negative distinguishing marks, extrapyramidal is differentiated from pyramidal rigidity, also by positive—for example, by cataleptiform phenomena, persistence of conditions of contraction in the musculature, fewness of movements, and so on. The whole clinical picture is

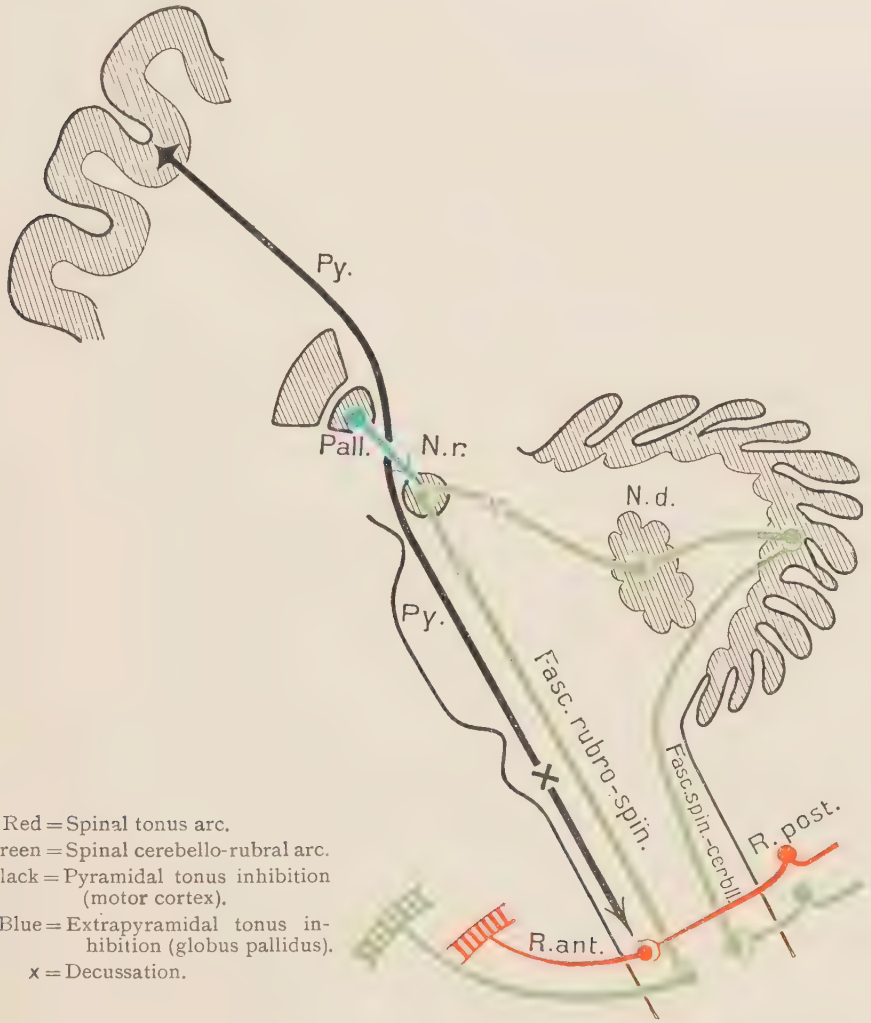


FIG. 98.—DIAGRAM ILLUSTRATING "PYRAMIDAL" AND "EXTRAPYRAMIDAL" RIGIDITY.

described as the *amyostatic symptom complex*, in contra-distinction to the *spastic* (*vide* Fig. 99). Whether the tremor—generally slow and rhythmical, occasionally taking characteristic forms, as in paralysis agitans—which often accompanies the amyostatic symptom complex is also a globus pallidus symptom appears questionable. A comparison of clinical, pathological, and experimental

findings tends to the conclusion that its ætiological connection is with the red nucleus.

Less certain than the relation of extra-pyramidal rigidity to lesions of the globus pallidus (*palæo-striatum*) is the dependence of choreic and athetotic phenomena on those of the *neo-striatum*—that is to say, the phylogenetically related putamen and caudate nucleus. So much is at any rate certain—namely, that chorea very often develops as a result of lesions of the mid-brain or its connections with the cerebellum without any demonstrable involvement of the corpus striatum. Further, no final answer has been given to the question whether choreic and athetotic phenomena may not be a symptom referable to the thalamus (*vide infra*, p. 193). With these qualifications, however, it is extremely probable that lesions of the neo-striatum may call forth the clinical pictures of chorea and athetosis, and of related torsion spasms of the type of *dystonia musculorum deformans*. Affections of the putamen probably tend rather to cause choreic, those of the caudate nucleus rather athetotic forced movements. There would seem to be a close relation between chorea and myoclonus, so far as the localization of the underlying anatomico-pathological condition in each case is concerned. The occasional combination of both syndromes, in the course of affections of the corpus striatum in epidemic encephalitis, renders this probable. All these hyperkinesias, it would seem, are to be regarded as symptoms of a removal of inhibition.

Fig. 99.—General ("Parkinsonoid") Muscular Rigidity in Bilateral Multiple Arterio-Sclerotic Tissue Degeneration (so-called "Lacunæ") in the Globus Pallidus.

In cases of symmetrical multiple softening lesions of both corpora striata, due to arterio-sclerosis (in which similar lesions are generally present in the optic thalami), a persisting incontinence of urine has occasionally been ob-

served, of the active or intermittent type described on p. 67. At approximately equal intervals, approximately similar quantities of urine are suddenly voided involuntarily in a jet. The power of voluntary micturition is lost, and the bladder is never emptied, there being always some "residual urine." The subcortical innervation of the bladder in the corpus striatum and the optic thalamus (*vide supra*, p. 67) is apparently bilateral.

We have already stated that thalamus lesions, if situated in the pulvinar, cause homonymous lateral hemianopia on the opposite side (p. 174); also that they usually lead, through action on neighbouring structures (nucleus caudatus and putamen), to hemiathetosis (exceptionally hemichorea), while the crossed partial hemiplegia frequently observed is undoubtedly a result of involvement of the internal capsule. The following, however, are to be regarded as direct symptoms of thalamus lesions:

1. *The abolition of certain psycho-reflexes.* Occasionally, for instance, the following paradoxic phenomenon is observed in thalamus lesions: In involuntary laughter or weeping, the lower facial muscles on the side opposed to the lesion remain masklike and immobile, the power to contract them voluntarily being at the same time quite unimpaired. As the opposite condition is sometimes found in capsular hemiplegias, the inference is justified that we have here to do with an interruption of a reflex arc as it passes through the thalamus.*

2. *A persistent crossed hemianæsthesia,* which is usually much less marked for tactile, painful, and thermal stimuli than for those connected with the *deep sensibility* ("muscular sense"). The interference with the latter causes *unilateral ataxia* and *stereo-anæsthesia.*

3. *Exceedingly severe and continuous pains,* occasionally exacerbated, and very refractory to analgesics. These *central pains* are referred to the contralateral half of the body, the hemianæsthesia alluded to above being thus given the impress of an *hemianæsthesia dolorosa* (*cf.* p. 30).

While the interference with psycho-reflexes above described points to a lesion in the anterior portion of the thalamus, the two last-named symptoms are more frequently observed when the lesion is situated in the posterior half of the organ. The character of the above-enumerated *direct thalamus symptoms,* whether they be of paralytic or of irritative type, corresponds with the function of the thalamus, the great connecting station, so to speak, through which practically the whole of the sensory tracts must pass before diverging to the sensory area in the cortex.

* Irritative lesions in the neighbourhood of this thalamic reflex centre may perhaps lead to "forced laughter" and "forced weeping."

III. SYMPTOMS DUE TO LESIONS OF THE HYPOPHYSIS.

Tumours of the hypophysis usually lead, in the first instance, and rapidly, to impairment of function in neighbouring nerves. The closely contiguous optic chiasma is particularly exposed to injury, and early disorders of the visual

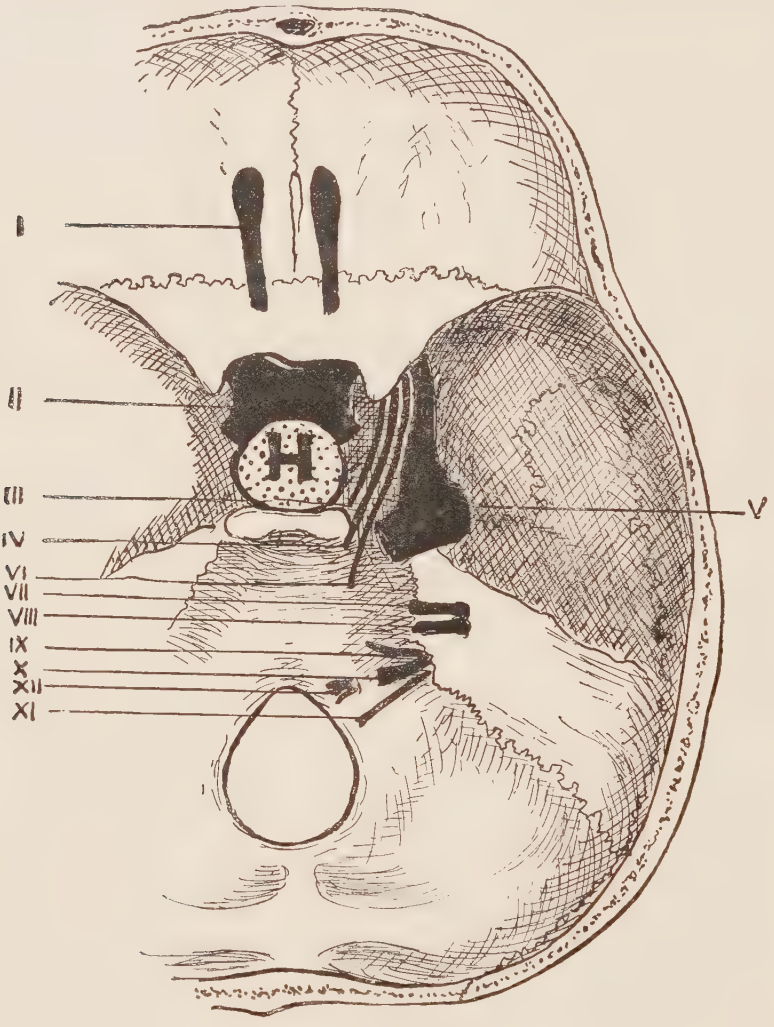


FIG. 100.—TOPOGRAPHY OF THE BASE OF THE SKULL.
H=Hypophysis. I-XII=Cranial nerves.

function are common. The most characteristic is *heteronymous bitemporal hemianopia*. There may, however, be double amaurosis from complete destruction of the chiasma, or unilateral amaurosis with hemianopia of the

other eye from unilateral destruction. Cysts of the hypophysis bulging laterally may cause nasal heteronymous hemianopia. The production of these symptom complexes has been discussed on p. 172 *et seq.* The nerves of the ocular muscles—oculo-motor, trochlear, and abducens—which pass close to the hypophysis on their way to the orbit, are very readily paralyzed by pressure from hypophysis tumours, as also is the first or ophthalmic ramus of the trigeminus (*vide* Fig. 100).

In many affections of the hypophysis (hæmorrhages, colloidal or fibrous degeneration, tumours), the symptom complex known as "acromegaly" is developed (enormous growth of the hands, feet, tongue, nose, and lower jaw). In other cases, however, there is evolved the condition known as "hypophyseal eunuchism" or "degeneratio adiposo-genitalis" (excessive growth of fat with arrested development of the genitals and of secondary sexual characters, such as pubic alteration of contour, axillary and pubic hair). It would seem that acromegaly is the result of a pathological over-stimulation of the hypophysis leading to an abnormal increase in the special internal secretion of the gland, while, on the other hand, degeneratio adiposo-genitalis is the expression of a secretory insufficiency on the part of the hypophysis. It must be borne in mind, however, that many cases of hypophysis tumours have been recorded in which neither acromegaly nor hypophyseal eunuchism was present. Detached adenomata of the hypophysis also (*e.g.*, in the sphenoid antrum) may lead to the development of acromegaly, while hypophyseal eunuchism may be produced by the action on the hypophysis of contiguous pathological processes in the base of the skull.

Further symptoms which, when the general signs of cerebral pressure are present, point to the hypophysis as the seat of the lesion, are: obstinate somnolence, polyuria with polydipsia (occasionally glycosuria), hydrorrhœa nasalis (intermittent flow of clear cerebro-spinal fluid through the nose). For a consideration of the question of Röntgen-ray diagnosis in this connection, *vide infra*, p. 198.

APPENDIX.

I.

CRANIO-CEREBRAL TOPOGRAPHY.

Cranio-cerebral topography is of the utmost importance in all operative procedures on the brain. In operative attacks on accessible morbid processes (superficial hæmorrhages, tumours, abscesses, etc.), the choice of the situation for trephining must, it need hardly be said, be based on the localization of the lesion, whatever it may be.

It is of enormous practical importance to be able to fix the position of the fissure of Sylvius and the central fissure on the outside of the cranial wall; for,

in the first place, cerebral surgery is, up to the present time, in the main a surgery of the central convolutions and the temporal lobe; and, in the second place, from the position of these two chief fissures the position of the others can be deduced without difficulty.

Fig. 101 gives the position of the cerebral fissures with reference to the external surface of the cranium. The two vertical lines seen in the figure, the so-called "pre- and post-auricular lines," are constructed as follows, with a view to the determination of the position of the two chief fissures: The former passes upwards from the anterior border of the external auditory meatus to the

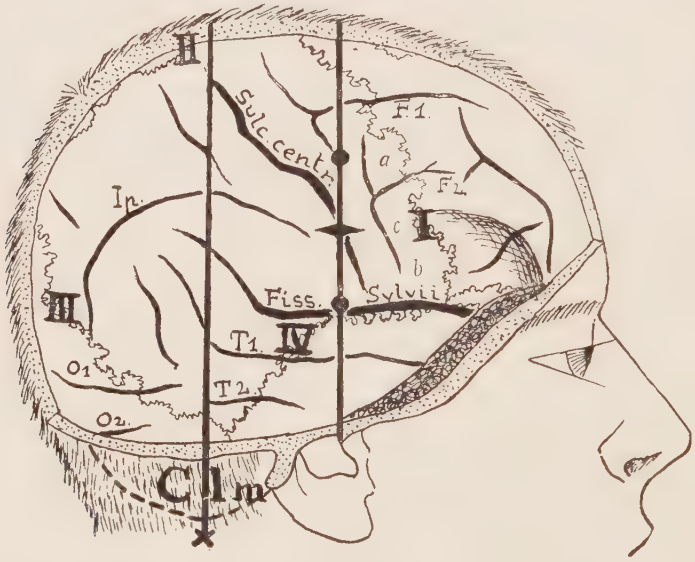


Fig. 101.—Projection of the Cerebral Fissures on the Surface of the Skull.

I = Coronal suture; II = Sagittal suture; III = Lambdoidal suture; IV = Temporal suture; F^1 and F^2 = First and second frontal fissures; O^1 and O^2 = First and second occipital fissures; Ip = Interparietal fissure; T^1 and T^2 = First and second temporal fissures; Clm. = Cerebellum; X = Posterior border of mastoid process.

vertex, being so drawn as to meet the sagittal line connecting the root of the nose and the occipital protuberance at right angles; the latter is drawn in an analogous manner, but from the posterior border of the mastoid process. The points *a* and *b* divide the pre-auricular line into three equal parts; *c* bisects it. If the trephine is applied between *c* and *b*, the lower end of the central convolutions will be reached, which in adults generally lies about 7 centimetres above the auditory meatus. The lower end of the central fissure often lies directly under the pre-auricular line. In our figure it is represented as reaching farther forward, for two types of relation between the brain and the outer cranial wall may be recognized—a *frontopetal* type, as represented in our figure, and an

occipitopetal type, in which the different fissures and convolutions lie rather farther back. The disposition of the brain is as a rule frontopetal in brachycephalic, occipitopetal in dolichocephalic individuals.

The post-auricular line crosses the upper end of the central fissure. The fissure of Sylvius will be met with about 1 to 1½ centimetres below the lower end of the central fissure. For some distance the Sylvian fissure lies immediately beneath the squamous suture. Under the parietal tuberosity lie, as a rule, the upper end of the fissure of Sylvius and the supramarginate gyrus.

An indication for prompt surgical interference is afforded by hæmorrhages

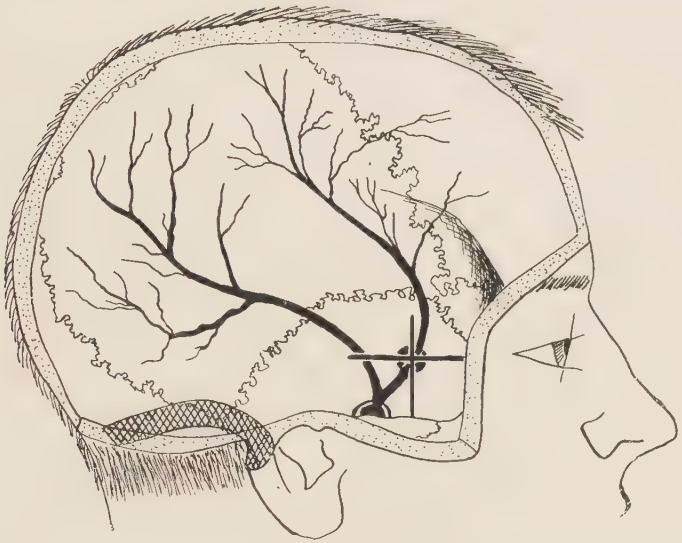


FIG. 102.—PROJECTION OF THE MIDDLE MENINGEAL ARTERY AND THE TRANSVERSE SINUS ON THE EXTERNAL SURFACE OF THE SKULL.

from the middle meningeal artery (after fractures of the skull, etc.). This vessel, a branch of the maxillary artery, enters the cranial cavity through the foramen spinosum, and divides into an anterior and a posterior branch. In order to ligature the common trunk, the trephine must be applied immediately above the middle of the zygomatic arch (*cf.* Fig. 102). The anterior branch is reached by trephining at the intersection of two lines, one of which is parallel to the zygomatic arch and two finger-breadths above it, while the other is a vertical line drawn to meet the first from a point just behind the zygomatic process of the frontal bone.

Fig. 102 shows the superficial projection of the transverse sinus, injury of which it is important to avoid in operations on the cerebellum.

II.

RÖNTGEN-RAY DIAGNOSIS.

The Röntgen rays play an important part in the localization of cerebral tumours. Direct Röntgenoscopy of the tumour is indeed impossible, unless the latter has undergone some calcification. Valuable conclusions may, however, be drawn from changes in the bones of the skull revealed by the rays.

The most significant of these changes are circumscribed areas of involvement of the cranial walls. Thus, tumours of the hypophysis often erode the *sella turcica*, those of the cerebello-pontine angle the posterior surface of the *dorsum sellæ*, new growths of the cerebellum, the squamous portion of the occipital, etc. On the other hand, circumscribed thickenings of the cranial wall originating from the dura mater are sometimes observed in the neighbourhood of tumours. Occasionally we have both thickening and erosion of bone substance. Again, a localized pushing out and thinning of the cranial wall may be noticed over the site of a tumour. Finally, the *Venæ diploeticæ*, which are normally hardly recognizable in Röntgen pictures, are occasionally so much distended in cases of cerebral tumour that they appear as broad bands on the plate, the distension being most marked in the neighbourhood of the tumour.

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